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Volume 30, issue 1

Canadian Medical Association

Volume 30, No. 1, January 1987

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Allografts of Bone
The Hartmann Procedure

The Canadian Journal of Surgery

Le journal canadien de chirurgie

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Reference: 1. Birdsell DC, Davidson JSD: A Report on Clinical Trials of NU GAUZE* Rayon Sponges, Including In Vitro and In Vivo Comparisons with Conventional Cotton Gauze Sponges. July 1986

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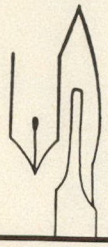
HISTORY OF SURGERY

QUILL ON SCALPEL

This section provides a medium through which Canadian surgeons can declare themselves, briefly and informally, on the day-to-day affairs of surgery.



Morbidity Audit, 1984, Canadian Society for Vascular Surgery

Participation in the annual morbidity audit of the Canadian Society for Vascular Surgery increased in 1984 as the concept of the audit became clearer and the feelings of the members towards the society became more positive. Most surgeons followed the directions carefully, using a single line for each patient with complications, thus making correlation of complications possible.

Individual results are confidential but allow the surgeon to follow his profile each year and to compare it with the national average. The national results continue to be very good (Fig. 1).[1] The calculation of "COMPLICATED" was changed in the 1984 audit so that death is not counted as a complication (i.e., morbidity and mortality are separated). Grafts are considered "FAILED" if they became occluded or there was a subsequent amputation.

Figure 2 summarizes the incidence of the selected complications before hospital death. These results include reported mortality from the 1983 and 1984 audits and a few "late entries" from 1983. The association of these complications with death is striking. This explains why good vascular surgeons are obsessive compulsive! The cause of death was recorded for 200 patients during 1983 and 1984 (Fig. 3). Ruptured aneurysm excluded, 67% of hospital deaths within 30 days were cardiac, which reflects our patient load.

Because of renewed interest in infrainguinal reconstruction, the 1986 audit will be extended in this area to include symptoms, repeat operations, outflow and in situ vein. Based on the encouraging participation in the audit, the Canadian Society for Vascular Surgery has embarked on a prospective study of aneurysmal surgery.

The executive of the society would like to thank the following members who contributed to the 1984 audit: F.M. Ameli, W. Atherton, H.H. Au, V.K. Balachandra, G.G. Barber, H. Basian, P.E. Blundell, G. Bondar, M.G. Bottomley, K.J. Bradley, P.M. Brown, R.T. Brownlee, S.E. Carroll, R.C.J. Chiu, J.C. Cole, G. DeRose, Y. Douville, A.R. Downs, R.S. Dunn, J.W. Dutton, E.T. French, P.D. Fry, A.N. Gerein, J.L. Gilmour, M.M. Goldbach, M.R. Goldberg, K.C. Grant, J.R. Gutelius, A.C. Heide, H. Hildebrand, W.G. Jamieson, K.W. Johnston, W.L.M. King, J. Lassonde, F. Laurendeau, B.J. Lawlor, G. Lehman, H.K. Litherland, C.K. Lye, G.D. Maddison, R. Maggisano, J.B. Marchuk, D. Marois, T.M. Maxwell, J.S. McGoey, N. McKenzie, G.E. Meads, A.H. Michalski, R.C. Moffat, V.B. Moonje, D.S. Mulder, F.G. Murphy, M.A. Naqvi, M.G.

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Detailed instructions to contributors, in English and French, appear on page 41 of this issue.

All prescription drug advertisements in the Journal have been precleared by the Pharmaceutical Advertising Advisory Board.

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AUDIT '84

Operation	Cases	COMPLICATED	(%)	Rev/T	Hg	Inf	FAILED	Amputation BK	AK	DIED	(%)
Aortic Aneurysm	952	49	(5)	3	7	13	2	2	3	31	(3)
Ruptured AA	259	101	(39)	3	5	4	1	2	3	96	(37)
Aortofemoral	924	67	(7)	16	4	20	10	3	6	20	(2)
Femorofemoral	370	42	(11)	12	5	12	12	4	4	11	(3)
Axillofemoral	140	30	(21)	3	2	2	16	5	4	10	(7)
Fem-pop vein	768	81	(11)	24	10	20	37	10	11	8	(1)
Fem-pop synth	598	75	(13)	38	4	10	36	13	11	3	(1)
Fem-tibial	273	65	(24)	21	2	6	35	22	12	4	(1)
Profundaplasty	272	22	(8)	7	3	3	6	2	4	3	(1)
Totals	4556	532		127	42	90	155	63	58	186	
			(%)					TND	Stroke		(%)
Carotid end'y	688	52	(8)	4	12	2	5	21	15 2.2%	8	(1.2)

(70 surgeons reported)

FIG. 1—This audit report is generated from audit form on personal computer and circulated to membership annually. COMPLICATED = one or more of following complications: Rev/T = revision and/or thrombectomy, Hg = hemorrhage (requiring operation), Inf = infection (wound, graft), BK = below knee, AK = above knee; TND = transient neurologic deficit.

Complications Associated With Mortality '83 & '84

Operation	DIED	Complicated	(%)	Rev/T	Hg	Inf	Fail	Amputated BK	AK
Aortic Aneurysm	66	12	(18)	3	5	2	3	1	1
Ruptured AA	175	12	(7)	6	6	1	1	1	0
Aortofemoral	40	11	(28)	2	0	4	3	0	5
Femorofemoral	15	6	(40)	1	0	0	7	0	3
Axillofemoral	24	6	(25)	1	0	2	4	0	3
Fem-pop vein	15	4	(27)	2	1	2	2	1	1
Fem-pop synth	18	7	(39)	4	1	2	4	3	2
Fem-tibial	11	3	(27)	2	0	0	3	1	1
Profundaplasty	5	3	(60)	0	1	0	2	0	1
								TND	Stroke
Carotid end'y	12	7	(58)	1	1	0	0	0	6

FIG. 2—Importance of surgical complication after vascular surgery is underlined by "Complicated" column. If deaths from ruptured aneurysm are eliminated, 28% of patients who died had one of selected complications before death.

CAUSE OF DEATH

Operation	Cardiac	Pulm	Renal	Multi organ	OR	Hg	Inf	DEATHS
Aortic Aneurysm	25	5	6	1	0	2	0	39
Ruptured AA	31	2	11	9	10	21	3	87
Aortofemoral	13	3	1	3	1	0	0	21
Femorofemoral	5	0	1	1	0	0	1	8
Axillofemoral	9	2	2	0	0	0	2	15
Fem-pop vein	8	1	0	0	0	0	0	9
Fem-pop synth.	9	0	1	0	0	0	1	11
Fem-tibial	4	0	0	0	0	1	1	6
Profundaplasty	0	1	0	0	0	0	0	1
Carotid end'y	3	0	0	0	1	0	0	4
Totals	107	14	22	14	12	24	8	201

FIG. 3—When deaths from ruptured aneurysm are eliminated, 67% of the hospital (30-day) deaths were from cardiac causes.

O'Dwyer, N.V. Perera, W. Pisesky, J.L. Provan, A. Rankin, R.H. Ritchie, R.H. Roy, T.K. Scobie, H.J. Scott, P.A. Shah, J.G. Sladen, J. Sweeney, J.F. Symes, P. Walker, R.D. Weisel, J.L. Wellington, L. Wooster.

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Reference

1. SLADEN JG: Morbidity audit, Canadian Society for Vascular Surgery. *Can J Surg* 1985; 28: 298-299

NOTICES

Applied Basic Science Course

"Behaviour of the Growth Plate" is the title of the 14th annual applied basic science course sponsored by the Division of Orthopaedic Surgery of the University of Ottawa, together with the Pediatric Orthopaedic Society of North America. The course will be held May 13–15, 1987 at the Health Sciences Complex; more information can be obtained by contacting Dr. Hans K. Uhthoff, Division of Orthopaedics, Ottawa General Hospital, 501 Smyth Rd., Ottawa, Ont. K1H 8L6; (613) 737-8377.

Extended Programs in Medical Education

The School of Medicine of the University of California is sponsoring a course entitled "Decisions in Surgery" Mar. 7–14, 1987, in Snowmass, Colo. The course is accredited for 20 hours in Category 1 by the American Medical Association and the Canadian Medical Association. Also being held is a "Postgraduate Course in General Surgery" Apr. 30–May 2, 1987, in San Francisco that is accredited for 15 hours in Category 1. For registration information for either of these courses call (415) 476-5808 and for program information call (415) 476-4251.

Postgraduate Education

Programs in colposcopy, gynecologic laser surgery and cutaneous laser surgery will be held Feb. 11–14, 1987, in San Diego, Calif., July 29–Aug. 1 in Quebec City and Oct. 25–29 in Bermuda. For more information write to Biomedical Communications, PO Box 224, Komoka, Ont. N0L 1R0 or call (519) 471-0300.

SURGEONS' UPDATE

What's new in surgery is the subject of this column. The short items are designed to let readers know who's doing what and why. Surgeons are interested in what other surgeons are doing in research, education, practice and administration. Surgery is a vibrant specialty, and, as its practitioners, you must be the source as well as the readers of this column.

Stroke Research to Be Focus of New Institute

A new institute, to house the work of more than 200 researchers focusing on stroke and aging, heart and circulation and immunologic disorders, opened in mid-September 1986.

Adjacent to the University of Western Ontario and University Hospital in London, the John P. Robarts Research Institute (Fig. 1) is a $25 million facility headed by H.J.M. Barnett, FRCPC. On hand for the opening was federal Health Minister Jake Epp who presented a cheque for $1.15 million as the latest instalment of the Canadian government's $4 million pledge and wished the staff "best of luck in the first 100 years". Ontario Premier David Peterson, London Mayor Tom Gosnell and ex-premier William Davis also joined the ceremonies.

Support for research in the institute's primary areas will come from personnel in clinical pharmacology, imaging and radiopharmaceuticals and clinical trials.

The setup for stroke research exemplifies the multidisciplinary approach planned; the team includes members of the divisions of neurology (Barnett, himself, and Vladimir Hachinksi), neurosurgery (S.J. Peerless), anesthesia (Adrian Gelb) and biophysics (Keith Farrar).

They will be investigating the fundamental mechanisms of cerebral ischemia and methods of "ischemic rescue": prevention of stroke after the atherosclerotic process has begun and minimizing the effects after stroke.

Said Syd Peerless, FRCSC, who chairs the division of neurosurgery, "The work on stroke is a major collaborative effort, and the institute is a unique facility, funded by provincial and federal governments as well as private donations [more than $11 million]; besides stroke, scientists will be investigating heart disease, aging, transplantation and clinical trials. These focuses are the strengths of the medical school. Each of the key areas has a separate floor but the researchers will share facilities and, hopefully, will enrich each other's work."

One of the first studies planned is an international cooperative comparison of medical and surgical prevention of stroke; the coordinators are Barnett and Peerless.

FIG. 1—Robarts Research Institute.

Contributions to this column are welcome. Please send your material to: Mrs. Amy Chouinard, *Canadian Journal of Surgery*, PO Box 8650, Ottawa, Ont. K1G 0G8.

A Study of Carotid Endarterectomy

More than 100 000 North Americans — of whom about 6000 are Canadians — undergo carotid endarterectomy annually to prevent stroke, and H.J.M. Barnett and S.J. Peerless, at the University of Western Ontario have designed a study to "confirm the value of the operation and identify patients most likely to benefit".

Attending a medical meeting in Hong Kong at the end of September, the two reported their plans for a multicentre project that will likely take in its first patients this summer. The protocol to compare the best medical treatment with the surgical intervention was finalized, and the cooperating centres — almost 50 scattered throughout this continent — have their respective ethics committees' approval. An application for a grant for funding has been submitted to the National Institutes of Health.

The overall budget is estimated at $15 million — an amount easily repaid by the elimination of unnecessary surgery if the study were to show that even a quarter of the operations are not indicated. "We expect there is enough interest for us to get the 3000 patients in 2 years," said Barnett.

Barnett and Peerless, who in 1985 published a study from 71 centres around the world, showed that extracranial–intracranial bypass did more harm than good. Peerless commented: "The bypass was a much smaller affair although it was becoming appreciable; endarterectomy has been done since the 1950s. So it's a standard procedure that's never been scientifically evaluated; diseases change so there's good reason, we think, to question whether it's effective."

Carotid endarterectomy has been the subject of increasing criticism, with a report from Toronto's Brian R. Chambers and John W. Norris recently being published in the *New England Journal of Medicine*. In Europe, where the operation is performed at a rate about half that here, scientists have launched their own study of its efficacy.

Canadian centres, besides the University of Western Ontario, collaborating in the Barnett–Peerless study are the universities of British Columbia, Manitoba, Toronto, Ottawa, Montreal, Laval, Dalhousie and McGill.

Symposium on Abdominal Surprises: 20 Questions

A 50-year-old man is explored for carcinoma of the right colon. Preoperative liver function test results are normal; however, at operation an unexpected 5-cm metastatic tumour is found in the

right lobe of his liver, deep and to the right of the gallbladder fossa.

Does the surgeon (a) resect the right colon and carry out a formal right hepatic lobectomy; (b) resect the right colon and leave the tumour in place, putting a catheter in the right hepatic artery through the gastroduodenal, for postoperative infusion of chemotherapeutic agents; (c) resect the right colon and leave the liver lesion for removal by right hepatic lobectomy in 3 to 6 weeks, after discussion with the patient; (d) resect neither tumour, treating the patient instead systemically with chemotherapy; or (e) resect the right colon and wait at least 6 months to see whether other metastatic disease appears before considering resection of the tumour in the liver?

The answer is (c) according to Robert M. Stone, FRCSC, and Bernard Langer, FRCSC, who prepared 3 of the 20 questions for this year's symposium on abdominal surprises — sponsored by the Canadian Association of General Surgeons at their annual meeting held in Toronto at the end of September.

Like last year, the symposium attracted more than 200 participants, and the group ran out of time before it covered all the questions about vascular, urologic and general surgical surprises. Copies of the questions, answers and commentary put together by the experts were available, with references cited from the recent literature on the topics. The event was the second annual, and the surgeons will probably have one again next year since it has generated such a good turnout and participation.

Cochairing the symposium were Joel Freeman, of the University of Ottawa, and Trevor Sandy, of the University of British Columbia. The latter spoke with CJS about the session: "The most sensational case was Marvin Wexler's (secretary of CAGS), the last one we had time for: a 30-year-old man, who had just returned from a 2-week stay in the Dominican Republic, had swallowed packets of cocaine, intending to smuggle them into the country. The packets became impacted and produced obstruction, but because they were only partially radiopaque, they were difficult to interpret on the x-ray films (Fig. 2). Eventually everyone realized that they were foreign bodies that had been ingested. The case stimulated a great deal of discussion.

"The urological cases were more practical, particularly the one on how to handle damage to the ureter during left hemicolectomy for carcinoma of the descending colon. The audience agreed with Michael Robinette's proposed method of dealing with the injury to minimize the risks of urinary leak during the postoperative period."

Robinette recommended repair immediately if the damage is recognized during surgery, but he preferred prevention by "preoperative intravenous pyelography or placement of ureteric catheters", especially in patients for whom some difficulty is anticipated. He commented that the ureteral "danger zones" are close to the inferior mesenteric vessels, the ovarian fossa, the rectovesical pouch, during division of the ligaments and during reperitonealization.

Said Sandy, "Dr. Stone dealt with the case of a perforated sigmoid encountered when one has made a small incision thinking that the problem is appendicitis. His recommendations, naturally, were that a new incision must be made and the perforated area removed by the Hartmann-type resection and exteriorization of the colon as a colostomy with plans to close it at a later stage."

Stone's presentation on carcinoma of the colon with isolated liver metastases generated considerable interest, said Sandy, because "it's now possible to do hepatic resections with a good anticipated survival in selected cases. However, it's inappropriate to do them when the metastases are discovered during surgery for the primary tumour."

T. Keith Scobie prepared questions about vascular injury; one was, in laparotomy for a gunshot wound to the abdomen, the discovery that the infrarenal inferior vena cava is transected and there are associated perforations of the colon and small bowel. The best method of management, he said, is ligation of the vena cava; however, if the suprarenal vena cava is injured, repair must be attempted.

Denis Bernard rounded out the group of experts; his questions included what to do with a 24-year-old woman who has Crohn's disease, has been taking steroids for 6 months, has lost 10 kg and has a tender pelvic mass. X-ray examinations show a narrowed ileum, an irregular area in the distal sigmoid colon but no fistula. At laparotomy the ileum is thick and inflamed, adhering closely to the sigmoid colon, which is also inflamed and indurated. An abscess between the ileum, the sigmoid and the right adnexa contains 50 ml of pus. The preferred management, he said, is to do resection anastomosis of the affected segment, establish ileostomy and mucous fistula and delay the proximal anastomosis, which still must be performed, to a second stage.

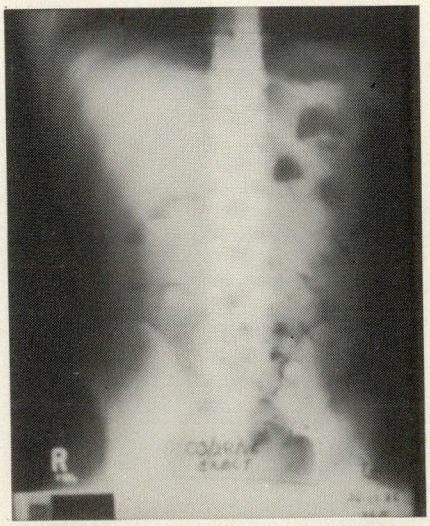

FIG. 2—Packets of cocaine that caused bowel obstruction.

CJS Coeditors Among Officers of American College of Surgeons

C. Barber Mueller, FRCSC, became second vice president-elect and Lloyd D. MacLean, FRCSC, was re-elected regent at the 72nd annual meeting of the American College of Surgeons last October.

Other fellows from Canada who were elected ACS governors at the meeting were Robert J. Bury (Edmonton), Alan D. Forward (Vancouver), S.E. O'Brien (Hamilton) and Andre Rioux (Quebec).

This month's *ACS Bulletin* carries presentations from a session on what's new in surgery, held Oct. 24 during the congress.

E.B. Tovee Named Honorary Member of General Surgeons

The Canadian Association of General Surgeons made Bruce Tovee, FRCSC, an honorary member Sept. 23 during its annual meeting and dedicated its scientific program to the retired surgeon who had formerly headed general surgery at the Toronto General Hospital and the University of Toronto. A founding member of the Association, Tovee was president and adviser during the early years.

New Appointments, University of Toronto

Effective in November, J. Kostuik, FRCSC, became head of the orthopedic division at Toronto General Hospital and Mount Sinai Hospital, replacing A. Gross, FRCSC.

R. Zuker, FRCSC, took up the position of head of plastic surgery at The Hospital for Sick Children.

AMY CHOUINARD

MARK E. BOYD, MD, FRCSC, FRCOG

Postoperative Gynecologic Infections

Postoperative gynecologic infections are caused by mixed aerobic and anaerobic bacteria that normally reside in the vagina. For the most part the infection, manifested by fever, resolves spontaneously, but occasionally serious problems result. Opinion now favours the prophylactic use of antibiotics before both abdominal and vaginal hysterectomy. Established infections are first treated with a combination of clindamycin and gentamicin.

Les infections gynécologiques postopératoires sont causées par une flore mixte aérobie et anaérobie qui réside normalement dans le vagin. Dans la plupart des cas, l'infection se manifeste par de la fièvre, cède spontanément mais, à l'occasion, de sérieux problèmes peuvent en résulter. L'opinion favorise actuellement l'usage préventif d'antibiotiques avant une hystérectomie abdominale ou vaginale. Les infections établies sont traitées en première intention avec une association de clindamycine et de gentamicine.

Hysterectomy is the most common major gynecologic operation and fever its most frequent complication. Usually the fever will resolve without antibiotic therapy or major ill effect; however, it is not entirely innocuous. The fever increases the patient's discomfort and anxiety, it may prolong hospitalization and occasionally is the forerunner of a serious disorder.[1]

The fever is generally from an infection at the operative site, particularly the vaginal vault and is caused by the bacterial inhabitants of the vagina. Our understanding of the process has advanced, with clarification of the bacteriologic characteristics of the vagina and the indications for and appropriate selection of antibiotic therapy. I shall first discuss the responsible bacteria and their antibiotic sensitivity, then the development of infection and its manifestations and finally the prevention of infection and the treatment of established infections.

From the Department of Obstetrics and Gynecology, Royal Victoria Hospital, Montreal, PQ

Accepted for publication Jan. 29, 1986

Reprint requests to: Dr. M.E. Boyd, Gynecologist-in-chief, Royal Victoria Hospital, Women's Pavilion, 687 Pine Ave. W, Montreal, PQ H3A 1A1

Bacteria and Antibiotic Sensitivity

Understanding this postoperative complication depends on an appreciation of the complex bacterial make-up of the vagina. Hysterectomy exposes the pelvic peritoneum, the soft tissues between it and the vagina, the cut edge of the vagina and the abdominal incision to the bacteria that are the natural habitants of the vagina and cervix. There is no single pathogen but a complex interrelated and interdependent group of bacteria (Table I); all types present are inoculated into tissue planes at the time of surgery. The interrelations between various groups are poorly understood, although their mutual dependence is such that the removal of one group adversely affects the metabolism of others. Postoperative infections are caused by *anaerobic bacteria (Bacteroides* sp., *Peptostreptococcus* sp., *Peptococcus* sp., *Clostridium* sp.) and the *aerobic bacteria* (*Staphylococcus epidermidis, Streptococcus* sp. [Lancefield groups A, B and D], *Gardnerella vaginalis, Lactobacillus* sp., *Escherichia coli* and *Proteus* sp.) of the vagina. We are especially concerned with the anaerobes, which are present in much larger numbers than the facultative bacteria (10:1).

Anaerobes

Two groups of anaerobes are present in almost equal numbers in the vagina — the gram-negative and the gram-positive anaerobes.[2] The predominant gram-negative anaerobe is *Bacteroides bivis*, which has an antibiotic sensitivity similar, but not identical, to *Bacteroides fragilis*. The anaerobic gram-positive organisms are represented by *Peptococcus* sp. and *Peptostreptococcus* sp.; *Clostridium* sp. is not an important member of this group.

Antibiotic therapy.—The gram-negative anaerobes are sensitive, in descending order, to each of the following antibiotics: clindamycin, chloramphenicol, metronidazole, cefoxitin and piperacillin. These drugs are also effective in dealing with the gram-positive anaerobes. Treatment, therefore, although directed toward the gram-negative organisms, is equally effective for the gram-positive ones. The reverse is not true. Although the gram-positive anaerobes are extremely sensitive to penicillin or any penicillin-like drug (cephalosporin), these drugs have limited value in the treatment of gram-negative infections.

Table I—Representative Bacteria Present in Vagina, Their Sensitivity

Bacteria	Antibiotic sensitivity
Aerobic	
Gram-positive	
Staphylococcus epidermidis, Streptococcus, groups A and B	Penicillin or cephalosporins
Streptococcus, group D	Ampicillin or piperacillin
Gram-negative	
Escherichia coli	Aminoglycoside, cefoxitin (2nd gen. cephalosporin), cefotaxime 3rd gen. cephalosporins) or moxalactam (3rd gen. cephalosporins)
Anaerobic	
Gram-positive	
Peptostreptococcus sp, *Peptococcus* sp, *Clostridium* sp	Penicillin, cephalosporin or piperacillin
Gram-negative	
Bacteroides bivis, B. disiens, B. fragilis, B. melaninogenicus	Clindamycin, chloramphenicol, metronidazole, cefoxitin, piperacillin

Clindamycin is the favoured antibiotic. It is especially effective in treating abscesses, in which anaerobic bacteria are present in large numbers. Its efficacy is related to its ability to enter polymorphonuclear leukocytes. It is thus transported into the abscess cavity and placed in contact with those bacteria that enter the leukocytes.[3] Metronidazole duplicates clindamycin's anaerobic coverage and is equally successful in penetrating the abscess cavities, but it does not provide the same extensive aerobic gram-positive and gram-negative coverage as clindamycin.[2]

Aerobes

The inoculum of aerobic bacteria is sparse compared with that of anaerobic bacteria. Streptococci and *E. coli* were formerly considered the primary pathogens in postoperative gynecologic infections. This is no longer the case, and they are now recognized as individual parts of the spectrum of bacteria involved.

Antibiotic therapy.—Aerobic bacteria are sensitive to a wide range of antibiotics and their sensitivity is such that one usually does not choose antibiotics with this group primarily in mind. Penicillin and the cephalosporins are effective. The third-generation cephalosporins, although effective for gram-negative aerobes, show reduced activity against the gram-positive organisms.

The Lancefield group D *Streptococcus* is of special concern relative to the resistance of subgroups (the group D enterococci, of which *Streptococcus faecalis* is a member). Although enterococci are seldom isolated — being present in only 5% of cases — they are resistant to many of the antibiotics frequently chosen. For this reason they may be the cause of failure in a patient who does not respond to the usual antibiotic therapy. They are sensitive to ampicillin and the extended penicillins (e.g., piperacillin).

The aerobic gram-negative bacteria, present in 10% of cases, are represented by *E. coli*. Therapy is directed toward this group because of their unique antibiotic sensitivities and they normally respond to aminoglycosides (i.e., gentamicin). Renal function is rarely compromised in gynecologic patients, and usually aminoglycosides may be safely prescribed. In the future, third generation cephalosporins (e.g., cefotaxime, moxalactam) may replace the aminoglycosides for this purpose.

Development of Infection

Despite all efforts at mechanical cleansing preoperatively, the vagina is never completely free of bacteria. Following inoculation of these bacteria into the operative site, a number of factors can lead to the easy establishment of infection. These include the exudation of fluid from the necrotic pedicles and traumatized peritoneum;[4] the presence of foreign bodies, such as sutures; impaired leukocyte function, as a result of hematoma formation;[4] and the capsular polysaccharide secreted by the *Bacteroides* that favours abscess formation.[5]

The infection may or may not follow the classic biphasic infection of Weinstein and associates.[6] In Weinstein's model, the aerobic bacteria cause acute peritonitis, which is followed by abscess formation due to anaerobic bacteria. In infections after hysterectomy, an abscess may form without preliminary aerobic peritonitis.[7]

A wide spectrum of infection of the vaginal vault is possible. It ranges from a minor infection of the cut surface of the vagina to a small collection of purulent material above the vagina and below the peritoneum or a large pelvic abscess contiguous with the vaginal vault and spreading into the peritoneal cavity.

An important variant of this process is the tubo-ovarian abscess. In almost all circumstances such an abscess follows vaginal hysterectomy in which the tubes and ovaries have not been removed. The cause of the abscess is inadequate hemostasis or infection of the ovary through its disrupted capsule.[8] Clinically, fever develops late (after 5 days) and a pelvic mass is normally present.[9]

An abscess may also develop in the abdominal incision. Its features are well known to the general surgeon. It can account for prolonged hospital stay and there may be a need for secondary surgery.

Another serious complication of pelvic infection is that of septic thrombophlebitis. The infection causes inflammatory changes in the veins of the pelvis and the production of heparinase; intravascular clotting of the pelvic veins may result.

Prophylaxis

General Measures

Febrile morbidity (oral temperature more than 38°C on two or more occasions, at least 6 hours apart during any consecutive 48-hour period, excluding the first 24 hours after operation) and infectious morbidity (clinical evidence of specific postoperative infections)[10] can be prevented to some extent by all of the following measures: meticulous surgical technique,[11] the replacement of catgut with polyglycolic acid sutures, delayed primary closure of the abdominal incision in obese patients or potentially infected cases,[12] the use of suction drainage from the vaginal vault[10] and leaving the vaginal vault open at the time of surgery.

Prophylactic Use of Antibiotics

Antibiotics given prophylactically have been shown to be effective in reducing both febrile morbidity and a number of serious postoperative sequelae (abscess or septic thrombophlebitis). Also, they are known to be safe, in that they do not mask pelvic infections or adversely change the vaginal flora. Increased numbers of anaerobic bacteria are found in the vagina after surgery, but this change in the vaginal flora is directly related to surgery not the use of antibiotics.[13]

Prophylactic antibiotics eliminate selected bacteria in the vagina, some of which contaminate the wound. The host's defence mechanisms will control the remainder that, as a result of a changed bacterial environment, will have been adversely affected. The antibiotic must be present in the tissues at the time of surgery; consequently, antibiotic therapy immediately preoperatively is the most important and often the only time it is needed.[14] The choice of antibiotic does not seem crucial. First-generation cephalosporins are excellent for the purpose; they are inexpensive and nontoxic. They are effective even when given as a single dose although gram-negative anaerobes and enterococci are resistant to these drugs.[14] The second-generation cephalosporin (cefoxitin) provides better gram-negative anaerobic coverage than the first-generation one. Cefoxitin has been used with considerable success as a single-dose prophylactic antibiotic before hysterectomy.[15]

Some years ago, when the prophylactic use of antibiotics was first introduced into gynecologic practice, opinion favoured use of the antibiotics at the time of vaginal hysterectomy but not with abdominal hysterectomy. At that time, complications after vaginal hysterectomy were more frequent and more serious than those after abdominal hysterectomy. Over the years, the prophylactic use of antibiotics with vaginal hysterectomy has been generally accepted and its effectiveness confirmed by 12 of 15 double-blind studies.[16] So successful is it that abdominal hysterectomy now has a higher febrile morbidity than its vaginal counterpart. Although individual surgeons are best able to judge the need for prophylaxis from their own experience, opinion now favours the prophylactic use of antibiotics with abdominal hysterectomy.[17] Striking reductions in febrile morbidity after abdominal hysterectomy are possible. A recent report[18] showed a reduction from 54% to 4%. The frequency of serious postoperative infections and length of hospital stay are also reduced.[18,19]

Treatment

If infection does become established,

different antibiotics should be prescribed. The choice is empiric, for one is often unable to obtain a proper culture. In anaerobic infection, antibiotic sensitivity is rarely helpful, but experience has shown that a combination of clindamycin and gentamicin is the first choice, and results are better if it is used immediately. Ampicillin or a first-generation cephalosporin is not recommended for established infections.[20] It is possible to replace the clindamycin/gentamicin combination with a single antibiotic. It could be a second-generation cephalosporin (cefoxitin), a third-generation cephalosporin (cefotaxime or moxalactam) or a ureidopenicillin (piperacillin); all are worthwhile in terms of both convenience and cost,[21] although none of them give better clinical results than clindamycin with gentamicin.[22]

In most circumstances, the clinical response to antibiotics is gratifying. If the response is not satisfactory the patient should be re-examined. There may be an infected hematoma with a bulging vaginal vault. Pelvic examination often results in the immediate drainage of foul dark material through the vaginal suture line, with the quick disappearance of the fever.

In the few remaining cases that have not responded to treatment, additional therapy is needed. In all, the antibiotic therapy should first be reviewed. If clindamycin with gentamicin has not been prescribed it should be tried. The failure to respond may be the result of resistant organisms, and additional antibiotic therapy is often directed toward enterococci. Thus, ampicillin or large doses of aqueous penicillin (60 million units daily) may be added to the clindamycin/gentamicin combination.

Surgery may be required to drain a pelvic abscess or remove a tubo-ovarian abscess. On clinical examination the presence of a pelvic mass may be obvious; ultrasonography will confirm the diagnosis.[23] Gallium radionucleotide scanning is used to locate an abscess that is not clinically apparent. An abscess does not always require surgery, as the antibiotics prescribed often result in spontaneous resolution. If there is no response within 48 hours surgical removal or drainage is indicated.

If the mass is contiguous with the vaginal vault, vaginal drainage is often possible; otherwise a midline abdominal incision should be made and the abdomen and the pelvis explored. Altered anatomy is first restored and a careful search made for multiple abscesses. If the adnexae are to be removed, do so after identifying the ureters and ligating the ovarian vessels. Generous peritoneal lavage with warm saline is performed, and Jackson-Pratt drains are used as required.

Finally, there are patients with no abscess cavities, who, despite proper antibiotic therapy, show no clinical response. In such cases, *septic thrombophlebitis* is the likely diagnosis. Despite a high fever and chills these patients often look well. Pelvic examination characteristically shows no abnormality. A notable response to heparin therapy confirms the diagnosis. Of further concern is the possibility of *ureteric obstruction* with resultant pyelonephritis. Injury to the ureter may result from the "easiest hysterectomy" and there may be few clinical findings to suggest such injury. The possibility must be considered, hence intravenous pyelography is part of the investigation of a gynecologic patient with unexplained fever postoperatively.

References

1. SWARTZ WH: Prophylaxis of minor febrile and major infectious morbidity following hysterectomy. *Obstet Gynecol* 1979; 54: 284–288
2. GALL SA, KOHAN AP, AYERS OM, et al: Intravenous metronidazole or clindamycin with tobramycin for therapy of pelvic infections. *Obstet Gynecol* 1981; 57: 51–58
3. KLEMPNER MS, STYRT B: Clindamycin uptake by human neutrophils. *J Infect Dis* 1981; 144: 472–479
4. HILL GB: Enhancement of experimental anaerobic infections by blood, hemoglobin, and hemostatic agents. *Infect Immun* 1978; 19: 443–449
5. BARTLETT JG, DEZFULIAN M, JOINER K: Relative efficacy and critical interval of antimicrobial agents in experimental infections involving *Bacteroides fragilis*. *Arch Surg* 1983; 118: 181–184
6. WEINSTEIN WM, ONDERDONK AB, BARTLETT JG, et al: Experimental intra-abdominal abscesses in rats: development of an experimental model. *Infect Immun* 1974; 10: 1250–1255
7. SWEET RL, YONEKURA ML, HILL G, et al: Appropriate use of antibiotics in serious obstetric and gynecologic infections. *Am J Obstet Gynecol* 1983; 146: 719–739
8. LEDGER WJ, CAMPBELL C, TAYLOR D, et al: Adnexal abscess as a late complication of pelvic operations. *Surg Gynecol Obstet* 1969; 129: 973–978
9. HEVRON JE JR, LLORENS AS: Management of postoperative abscess following gynecologic surgery. *Obstet Gynecol* 1976; 47: 553–556
10. KVIST POULSEN H, BOREL J: T-tube suction drainage and/or prophylactic two-dose metronidazole in abdominal hysterectomy. *Acta Obstet Gynecol Scand* 1984; 63: 711–714
11. RICHARDSON AC, LYON JB, GRAHAM EE: Abdominal hysterectomy: relationship between morbidity and surgical technique. *Am J Obstet Gynecol* 1973; 115: 953–961
12. BROWN SE, ALLEN HH, ROBINS RN: The use of delayed primary wound closure in preventing wound infections. *Am J Obstet Gynecol* 1977; 127: 713–717
13. OHM MJ, GALASK RP: The effect of antibiotic prophylaxis on patients undergoing vaginal operations. I. The effect on morbidity. *Am J Obstet Gynecol* 1975; 123: 590–596
14. HAMOD KA, SPENCE MR, ROSENSHEIN NB, et al: Single-dose and multidose prophylaxis in vaginal hysterectomy: a comparison of sodium cephalothin and metronidazole. *Am J Obstet Gynecol* 1980; 136: 976–979
15. HEMSELL DL, HEMSELL PG, HEARD ML, et al: Preoperative cefoxitin prophylaxis for elective abdominal hysterectomy. *Am J Obstet Gynecol* 1985; 153: 225–226
16. SWEET RL, GIBBS R: *Infectious Diseases of the Female Genital Tract*, Williams & Wilkins, Baltimore, 1985
17. Prophylactic use of antibiotics with abdominal hysterectomy. *American College of Obstetricians and Gynecologists Newsletter* 1985; 29: 1–12
18. ROBERTS JM, HOMESLEY HD: Low-dose carbenicillin prophylaxis for vaginal and abdominal hysterectomy. *Obstet Gynecol* 1978; 52: 83–87
19. HOLMAN JF, MCGOWAN JE, THOMSON JD: Perioperative antibiotics in major elective gynecologic surgery. *South Med J* 1978; 71: 417–420
20. LEDGER WJ: Selection of antimicrobial agents for treatment of infections of the female genital tract. *Rev Infect Dis* 1983; 5 (suppl 1): S98–104
21. RAMPHAL R: The third generation cephalosporins in obstetrics and gynecology. *Infectious Disease Letters for Obstetrics and Gynecology* 1983; 5: 13–18
22. SWEET RL, OHM-SMITH M, LANDERS DV, et al: Moxalactam versus clindamycin plus tobramycin in the treatment of obstetric and gynecologic infections. *Am J Obstet Gynecol* 1985; 152 (7 pt 1): 808–817
23. TAYLOR KJ, WASSON JF, DE GRAAFF C, et al: Accuracy of grey-scale ultrasound diagnosis of abdominal and pelvic abscesses in 220 patients. *Lancet* 1978; 1: 83–84

SESAP V Question

103. An awake and alert 46-year-old man has generalized abdominal pain after an automobile accident. Marked tenderness and involuntary muscle spasm are noted in the right upper abdominal quadrant. Blood pressure is 90/50 and pulse rate is 120/min. Chest roentgenogram, hematocrit, and urinalysis are normal. Which one of the following tests would be mandatory before a decision to perform an exploratory celiotomy is made?

(A) Peritoneal lavage
(B) Selective abdominal arteriography
(C) Computed tomography of the abdomen
(D) Abdominal sonography
(E) None of the above

For the question above, select the one answer that is best of the five given. For the critique of Item 103 see page 65.

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Abdominopelvic Computerized Tomography and Open Peritoneal Lavage in Patients With Blunt Abdominal Trauma: a Prospective Study

This prospective trial compares abdominopelvic computerized tomography and open peritoneal lavage in the diagnosis of blunt abdominal trauma.

Fifteen patients (group 1) were evaluated by both methods. Another 15 patients (group 2) had only computerized tomography. Criteria for a "positive" scan were hemoperitoneum and evidence of solid organ injury. Criteria for "positive" lavage were a grossly bloody return, erythrocyte count greater than 20.0×10^9/L and leukocyte count greater than 0.5×10^9/L. At laparotomy, only injuries requiring repair or excision were considered "true positive". Patients who did not have laparotomy and had an uncomplicated clinical course were considered "true negative". With tomographic criteria alone for diagnosis there would have been one false-positive and three false-negative results, compared with three false positive and no false negatives for open peritoneal lavage alone. None of the three patients who had negative findings on laparotomy suffered any morbidity or died. Results of computerized tomography and open peritoneal lavage agreed in 8 of 15 patients ($\varkappa$ value = 0.52), indicating a low level of agreement between the two.

The authors believe that open peritoneal lavage remains the diagnostic procedure of choice in blunt abdominal trauma.

On a comparé dans cette étude prospective les résultats de la tomographie axiale abdominopelvienne par ordinateur et ceux du lavage péritonéal dans le diagnostic des contusions abdominales.

Quinze malades (le groupe 1) ont été évalués par les deux méthodes. Quinze autres patients (le groupe 2) ont passé la tomographie seulement. Les critères d'une tomographie "positive" étaient la présence d'un hémopéritoine et la découverte d'une lésion à un organe solide. Les critères d'un lavage "positif" étaient la récupération d'un liquide de lavage sanguinolent, un décompte érythrocytaire supérieur à 20.0×10^9/L et un décompte leucocytaire supérieur à 0.5×10^9/L. A la laparotomie, seules les blessures nécessitant réparation ou excision ont été jugées "vraiment positives". Les patients n'ayant pas eu de laparotomie et dont l'évolution clinique n'a pas été compliquée ont été considérés "négatifs vrais". En s'appuyant sur les seuls critères tomographiques on aurait eu un faux positif et trois faux négatifs. Le lavage péritonéal seul aurait révélé trois faux positifs et aucun faux négatif. Aucun des trois malades chez qui la laparotomie a été négative n'a subi de morbidité ou n'est décédé. On a obtenu concordance des résultats de la tomographie axiale et du lavage péritonéal dans 8 cas sur 15 ($\varkappa$ = 0.52) ce qui indique une faible corrélation entre les deux techniques.

De l'avis des auteurs, le lavage péritonéal demeure le procédé diagnostique de premier choix dans les cas de contusions abdominales.

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The evaluation of abdominal injury resulting from blunt trauma remains a controversial issue. Abdominal injury must be considered in all patients with multiple trauma since it is common and potentially life-threatening. However, inappropriate interventions may detract from the management of other serious injuries and can create their own morbidity. Since clinical abdominal examination is not always reliable in this situation, other diagnostic methods have been developed and refined. They include peritoneal lavage and noninvasive procedures such as ultrasonography, computerized tomography (CT) and radionuclide studies.

Currently, the most widely used technique is peritoneal lavage. A number of reports have described the use of CT scanning to assess abdominal injuries.

Sunnybrook Medical Centre is an active trauma centre handling a large volume of patients with blunt trauma. Our interest in abdominal injury led us to compare open peritoneal lavage and CT scanning in the evaluation of blunt abdominal injury. We examine the preliminary results and some of the technical problems to be considered in obtaining good quality scans.

Patients and Methods

Patients with multiple-system trauma whose abdominal injury would normally be assessed by open peritoneal lavage were selected for study. Excluded were

patients who were hemodynamically unstable or had other obvious indications for immediate surgical intervention. Those who were hemodynamically stable and had no alteration in their level of consciousness were also excluded, since most of these patients can be followed up clinically. Fifteen patients (group 1) between the ages of 15 and 75 years were studied by both CT scanning and open peritoneal lavage.

After initial resuscitation, abdominopelvic CT scanning was performed on a GE CT/T 9800 scanner (General Electric Co., Unionville, Ont.), taking 15-mm cuts from the dome of the diaphragm to the symphysis pubis. A scan was judged to be positive (Figs. 1 and 2) if there was evidence of hemoperitoneum or solid-organ injury. Figure 1 shows evidence of extensive subcapsular hematoma of the liver with hemoperitoneum, and Fig. 2 shows free fluid (blood) in the pelvis.

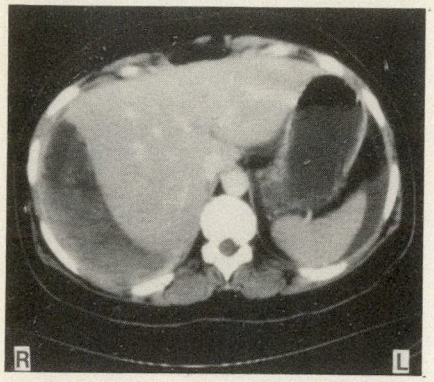

FIG. 1—Computerized tomogram of patient with ruptured subcapsular hematoma of liver with blood around liver and spleen.

The patient was then returned to the trauma room or to the operating room where peritoneal lavage was carried out by the open technique, through a vertical infraumbilical incision to visualize the peritoneum directly while maintaining strict hemostasis. The criteria for a positive lavage were: a grossly bloody aspirate, lavage erythrocyte count more than 20.0×10^9/L and leukocyte count more than 0.5×10^9/L. These criteria are in use at the regional trauma unit and are based on experience at this centre.[1] The patient was then managed appropriately by laparotomy or observation.

At laparotomy, only injuries requiring repair or excision gave a result that could be classified as true positive. Patients with a negative result from open peritoneal lavage did not have a laparotomy. When a patient had smooth recovery after observation the result of open peritoneal lavage was considered true negative.

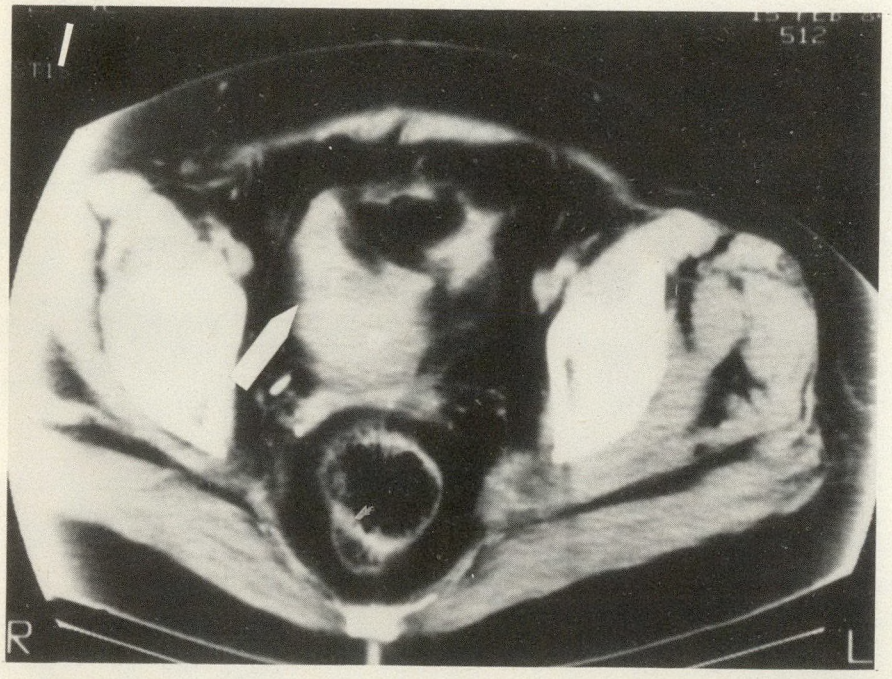

FIG. 2—Presence of free blood in pelvis (arrow) indicates intraperitoneal hemorrhage.

Table I—Results

Method	Positive	Negative	False positive	False negative
Group 1—decision based on				
CT alone	2	13	1	3
OPL alone	7	8	3	0
Group 2	4	11	3	0

Group 1 = 15 patients assessed by both computerized tomography (CT) and open peritoneal lavage (OPL). Group 2 = 15 patients assessed by CT only.

Results (Table I)

The average injury severity score of the 15 group 1 patients was 26. Computerized tomography in isolation gave one false-positive scan and three false-negative ones. In reviewing the false-negative scans in retrospect, the radiologist identified certain technical limitations, which included scatter artifact, failure to use contrast materials and incomplete scans.

Open peritoneal lavage in isolation gave three false-positive results but no false-negative ones. These patients had hemoperitoneum but suffered no organ injury requiring repair or excision. No patient suffered morbidity from a laparotomy that disclosed no organ injury requiring repair.

The results of the CT scan and open peritoneal lavage agreed in 8 of the 15 patients. The agreement between the two methods gave a κ value of 0.52, indicating a low level of agreement.

We also have data on 15 patients who underwent CT scanning only (group 2) to evaluate the abdomen for injuries. Their average injury severity score was 15 (range from 5 to 29). The same criteria for interpreting the scans and judging the outcome as true positive or negative were used. Four of these patients were considered to have a positive scan. One scan was judged to be true positive (a bleeding liver hematoma) and three false positive. These were from one patient with a retroperitoneal hematoma, another with a nonbleeding splenic tear and one who did not have a laparotomy but made a smooth recovery. Eleven other patients were thought to have a negative scan; none underwent laparotomy and all had an uncomplicated recovery (no false negatives).

Discussion

Abdominal injury continues to account for a large proportion of lethal and potentially lethal injuries resulting from blunt trauma. Sound judgement is required to determine which patients should have a laparotomy. The surgeon wants to avoid missing any serious injuries while not subjecting patients to inappropriate interventions that are potentially harmful and may detract from the management of other serious injuries.

Routine laparotomy plays no role in modern trauma care.[2] Clinical examination of the abdomen in patients with multiple trauma is unreliable during the initial period after injury or when there is any alteration in the level of consciousness. In a prospective study[3] of clinical examination and peritoneal lavage, a

definite positive or negative diagnosis could be made in only 58 (40%) of 142 patients. Of 23 patients with clinically positive abdominal findings, only 10 (43%) had important injuries. Of 35 patients with negative clinical findings, 12 (34%) had important injuries. Overall the clinical diagnosis was correct in 34 (59%). In this same series[3] of patients the accuracy of open peritoneal lavage was reported as 90%. Similar conclusions were reached in another prospective study[4] of physical signs in blunt abdominal trauma. Of patients who eventually had negative peritoneal lavage, bowel sounds were absent in 28% and guarding and rigidity were present in 20%. Of those eventually found to have a positive lavage, abdominal distension was absent in 66% and guarding and rigidity in 30%. It is clear that physical signs of abdominal injury cannot be relied upon in traumatized patients, particularly when there is any alteration in the level of consciousness.

Diagnostic tests to evaluate abdominal injury have been developed and refined during the past 15 years. Peritoneal lavage is an invasive test that has been used widely in evaluating blunt abdominal trauma. The open technique has been shown[5,6] to be superior to the percutaneous trocar technique with respect to potential iatrogenic injury as well as false-positive results. The test has its limitations. A false-positive lavage may result from poor technique with inadequate hemostasis or in cases with pelvic fractures and retroperitoneal hematoma. Open peritoneal lavage is not useful in patients with multiple abdominal scars or extensive adhesions. Certain injuries such as rupture of the diaphragm or of the retroperitoneal duodenum may be missed. With proper technique, sensitivity and specificity of approximately 98% can be expected.[1,7] This figure will depend partly on the cell count used as a criterion for a positive result.

A number of noninvasive radiologic methods have also become popular in the diagnosis of abdominal injury. They include abdominal ultrasonography, radionuclide scanning and abdominal CT scanning.[8] Since many patients with multisystem trauma will require CT scanning of the head to rule out head injury, CT scanning of the abdomen has been adopted by some trauma centres. The advantages of this technique are that it is noninvasive and detects retroperitoneal injuries. Some advantages in the evaluation of intraperitoneal trauma have also been claimed.[9] The largest experience with abdominal CT scanning for trauma has been reported from San Francisco.

This study is a more-formal attempt to assess the two diagnostic tests. In the preliminary phase we were hoping to establish the feasibility of CT scanning in the acute situation at our centre and to establish criteria for minimum technical quality of scans.

In assessing the value of a diagnostic test, the entire process of obtaining, interpreting and acting upon the results of that test is taken into consideration. Thus, if in the emergency situation, poor-quality scans are produced and they provide inadequate or incomplete information to make a diagnostic decision, these scans cannot be excluded from consideration but must be interpreted as a disadvantage of the technique. During this study we encountered several cases in which the scan gave misleading or incorrect diagnostic information because the technical quality was inadequate. In our three false-negative cases, technical problems were identified that, when viewed in retrospect, made the scan result inconclusive. In one of the cases, a liver laceration was missed because of scatter artifact at an interface with sharp contrasts. This made visualization of the liver parenchyma difficult. Two other injuries were missed because sufficient time was not taken to obtain complete scans from the dome of the diaphragm to the symphysis pubis. In one case an injury to the spleen requiring splenectomy was missed. In the other case a bowel perforation was missed. While this would not be diagnosed directly by CT scanning, evidence of fluid in the pelvis or subphrenic spaces might have

been visualized had a complete scan been done.

Had a treatment decision been made on the basis of the CT scan alone, there would have been one false-positive and three false-negative results. Had treatment decision been made from the results of open peritoneal lavage alone, three false-positive and no false-negative cases would have resulted. The other group of patients had only CT scanning to evaluate the abdomen. Three false-positive and no false-negative results were obtained.

These observations suggest that open peritoneal lavage is superior to abdominal CT scanning in the diagnosis of blunt abdominal trauma. The results are influenced by the technical limitations that were encountered with the CT scans. Technical problems were not encountered with open peritoneal lavage since this technique has been in use for some time at Sunnybrook Medical Centre. Just as attention to detail is essential to the successful performance of peritoneal lavage, so attention to technical detail and expert interpretation are necessary for the production of good-quality, reliable CT scans in trauma.

The technical problems encountered during the course of this study were due to the following:

(a) The uncooperative patient producing movement artifact. It may be necessary to restrain or sedate the patient. General anesthesia is rarely required or justified in order to obtain a CT scan.

(b) Incomplete scans. Fifteen-millimetre cuts must be taken from the dome of the diaphragm to the symphysis pubis.

(c) Failure to administer contrast materials. Contrast material must be administered intravenously just before scanning unless there is a clear history of allergy. Gastrointestinal contrast material should be administered approximately 20 minutes before scanning. No complications have arisen because of the use of gastrointestinal contrast material.

(d) Inadequate scanning equipment. The use of high-quality scanning equipment will eliminate some of the scatter artifact.

Conclusions

In this small preliminary study open peritoneal lavage was superior to CT scanning. We believe it remains the diagnostic modality of choice for evaluation of blunt abdominal trauma. Further studies of CT scanning for trauma with strict adherence to technical details are planned at our centre. We hope to define the role of both CT scanning and open peritoneal lavage in the evaluation of the traumatized patient.

References

1. McLellan BA, Hanna SS, Montoya DR, et al: Analysis of peritoneal lavage parameters in blunt abdominal trauma. *J Trauma* 1985; 25: 393–399

2. Bivins BA, Sachatello CR: Diagnostic exploratory celiotomy: an outdated concept in blunt abdominal trauma. *South Med J* 1979; 72: 969–970

3. Bivins BA, Sachatello CR, Daugherty ME, et al: Diagnostic peritoneal lavage is superior to clinical evaluation in blunt abdominal trauma. *Am Surg* 1978; 44: 637–641

4. Rodriguez A, DuPriest RW jr, Shatney CH: Recognition of intra-abdominal injury in blunt trauma victims. A prospective study comparing physical examination with peritoneal lavage. *Am Surg* 1982; 48: 457–459

5. Pachter HL, Hofstetter SR: Open and percutaneous paracentesis and lavage for abdominal trauma: a randomized prospective study. *Arch Surg* 1981; 116: 318–319

6. Moore JB, Moore EE, Markovchick VJ, et al: Diagnostic peritoneal lavage for abdominal trauma: superiority of the open technique at the infraumbilical ring. *J Trauma* 1981; 21: 570–572

7. Alyono D, Morrow CE, Perry JF jr: Reappraisal of diagnostic peritoneal lavage criteria for operation in penetrating and blunt trauma. *Surgery* 1982; 92: 751–757

8. Jones TK, Walsh JW, Maull KI: Diagnostic imaging in blunt trauma of the abdomen. *Surg Gynecol Obstet* 1983; 157: 389–398

9. Federle MP: Computed tomography of blunt abdominal trauma. *Radiol Clin North Am* 1983; 21: 461–475

SHENG CHIH-YONG*

Guest Lecture: Surgery in China Today

The fusion of traditional and modern concepts is a unique feature of medical practice in China. Since 1949, there have been considerable achievements particularly in the fields of burns, surgery of the liver, pancreas and the heart, and in microsurgery and limb reimplantation.

Surgical skills are now distributed through medical units outside the large cities, and at the grass-roots level, making them available to peasants. Research facilities remain limited and are responsible for the slow and uneven progress in surgery but the new China will spare no efforts to fill the gap and learn from all developed countries.

La fusion de concepts traditionnels et modernes est une caractéristique unique de la pratique médicale chinoise. Depuis 1949, on a assisté à des réussites importantes dans les domaines des soins aux brûlés, de la chirurgie hépatique, pancréatique et cardiaque, de la microchirurgie et des réimplantations de membres.

La compétence chirurgicale se retrouve maintenant dans les services médicaux situés hors des grandes villes, au niveau de la masse, et accessible aux paysans. Les installations de recherche demeurent réduites ce qui est responsable de développements lents et irréguliers en chirurgie. Néanmoins, la nouvelle Chine n'entend épargner aucun effort pour rattraper les retards et apprendre de toutes les nations développées.

In old China, surgery was practised only in large cities like Shanghai, Peking, Tientsin and Canton, and there were almost no facilities in the rural areas. In contrast, since the founding of the new China in 1949, there have been many achievements in surgery, not only advances in clinical surgery and the opening up of new fields, but also the widespread practice of surgery in medical units at the grass-roots level, making it more readily available to peasants. Chinese surgeons participate in the work of mobile medical teams, performing operations in county hospitals, commune health centres and even cooperative medical stations in production brigades. Mobile medical teams have also trained local medical personnel, enabling them to give surgical treatment for minor ailments. Following this extensive initial program, abdominal operations, such as partial gastrectomies, could be performed with success in most of the county hospitals and, in some, more complicated operations such as reimplantation of severed limbs and fingers, partial hepatectomy, partial pancreatectomy and shunt operations for portal hypertension.

One young surgeon, a graduate of Shanghai Medical College who now works in a small county in Henan, reported on 55 reimplantations of limbs with excellent results. Some surgeons have participated in research on cancer and schistosomiasis. The schistosomiasis groups, working in hyperendemic regions, have performed splenectomies for chronic disease with splenomegaly, and thus enabled thousands of seriously disabled patients to return to agricultural work. For instance, a team sent across the country by the First Medical College of Shanghai performed 1791 splenectomies in 10 years and reported an operative survival rate of 94.1%.

Burns

Since 1958, we have made great strides in the field of burn management, and it has gradually developed into an independent surgical discipline. At present there are at least 7 burn centres and over 30 burn units, with adequate facilities and expertise, all over the country. In many hospitals, however, burns are still managed by general surgeons.

The overall survival rate of patients with burns of varying degrees of severity has been as high as 95%, with the LD50 (lethal dose for 50% mortality) reaching 76%, and the LD50 for full-thickness burn 46.2%. In certain hospitals the results of burn treatment are even better, with the LD50 ranging from 80% to 87% and that of full-thickness burn as high as 52%.

Major burn wounds are exposed to air. Eschars of full-thickness burns are removed in stages after the patient's general condition stabilizes. Deep second-degree burns are often excised tangentially. The resulting wounds are then completely covered with sheets of allograft, in which there are previously cut small windows, 1 cm apart. Two days after grafting, small autografts are applied through the windows. If it has not been injured, the usual donor site for harvesting autograft is the scalp. Provided the wound excision is complete and the distance between autografts kept to 1 cm, the allograft will adhere to the wound for 3 weeks. When rejection occurs, it usually takes the form of desquamation, because the small autografts have grown and coalesced. It cannot be overemphasized that complete coverage of the wound with viable autografts and allografts is the single most important principle conducive to success of treatment.

We consider the scalp the best donor site for the following reasons: scalp epidermis grows rapidly, so wounds will heal in 5 to 7 days enabling us to harvest skin from the scalp as many as 22 times during the course of treatment; infection of the donor site is extremely rare; when the hair grows back, and as it usually grows more exuberantly than before, the scars are invisible.

The combined allogenic and autogenic skin grafting of the excised wound has been studied in depth. After 21 days, buds of autograft epithelium are seen to meet each other in between the superficial and deep portions of the dermis, creating the so-called "sandwich phenomenon". The epithelial cells seem to possess a much more intense immunologic antigenicity than fibroblasts, probably the reason why the rejection reaction first appears in the epithelial and superficial dermal layers — where more epithelial elements are present.

Excision of eschar when present is also indicated when septicemia occurs as a result of wound infection. In a report of 32 cases, septicemia was controlled after eschar excision in 18, with 17 survivors.

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With the advent of microsurgery, advances have been made in the management of electrical contact burns, especially in the forearm and cranium. In 1981, a group of burn surgeons in Beijing reported 14 cases in which vessel transplantation was done after early débridement for electrical burns of the wrist; nine hands were saved, with partial restoration of function. Residual defects were repaired by transplantation of free musculocutaneous or free omental flaps, with satisfactory results.

Electrical burn of the cranium may be treated likewise. Necrotic scalp is excised, the underlying superficial layer of the outer table of the skull is chiselled off and the wound closed, either by a rotating scalp flap or a free skin or omental flap, anastomosing the attached vessels with superficial temporal vessels. The hospital stay has been considerably shortened, the symptoms attributable to cranial defects have been greatly diminished and cosmetic results are good. In a recent study it was demonstrated that the apparently dead cranium could regenerate under coverage of a flap of tissue with adequate blood supply.

Research is encouraged and carried out in many institutes. Accomplishments in two major fields are worth mentioning. A group in Chongquid, Sichuan made a comprehensive study of inhalation injury, both in canine and goat models. They believe, contrary to what has been advocated, that fluid resuscitation should not be restricted because there is additional loss of fluid resulting from pulmonary injury. Restoration of adequate circulating blood volume is of primary importance in combating shock, and poor perfusion of lung tissue may itself induce increased capillary permeability and escape of fluid.

Infection is the second area of research where we pooled our efforts. In 87.5% of skin burn wounds, the organisms grown from the patient's blood are identical to those found in the wounds. However, early septicemia, occurring after serious shock may not be caused by organisms originating from the wounds. Most of the causative organisms in such cases are gram-negative rods and it has long been suspected that they take their origin from the intestinal tract. Recently, by the use of immunofluorescent staining, it was proven that *Pseudomonas aeruginosa* can traverse the intestinal wall. This finding strongly suggests the need for prophylactically administered antibiotics, especially when initial resuscitation is inadequate.

It has recently been demonstrated that anaerobes, like *Bacteroides*, play a role in burn wound infection but rarely invade the blood stream. Fungal infections, especially *Mucor* and *Phycomycetes*, have attracted attention, and, to facilitate early diagnosis, special techniques have been developed. Many clinicians are studying immunologic profiles in patients suffering major burns. We have arrived at the same conclusion, that in patients with major burns immunodepression involves nearly all facets of the host-defence mechanism.

It has been found that the chemoluminescent intensity of the neutrophil and the plasma fibronectin level may be used as predictors of sepsis. For immunotherapy, anti*Pseudomonas* immune human plasma and immunoglobulin have been developed. One group of surgeons claim that immune-RNA is efficacious in the treatment of burn sepsis.

Surgery

Liver

In China, primary hepatic carcinoma is relatively common. In recent years, the use of the α-fetoprotein test for mass screening of the population in regions with a high rate of hepatic cancer has resulted in the detection of many early small cancers.

Hepatoangiography, injecting radiopaque substances into the umbilical vein or hepatic artery, has been found in 86.4% of cases to localize small tumours with accuracy. Similarly, B-scan ultrasonography provides valuable information for the localization of small hepatocellular carcinoma giving a 50% accuracy rate. Early discovery and accurate localization are possible with early surgery and the postoperative survival rate is increased. In a report of 30 partial hepatectomies for early small cancers (diameter less than 5 cm), the 1-year, 2-year, 3-year and 5-year survival rates were 79.3%, 70.5%, 62.4% and 56.2% respectively compared with 8.4% to 23.6% 5-year survival reported in the literature. As a result of our experience in managing small hepatocellular carcinoma some surgical principles had to be revised. With a small hepatocellular carcinoma in the right lobe, a local resection instead of a lobectomy increases the resectability and decreases operative mortality. Bloodless hepatectomy (vascular isolation and hypothermic perfusion) may improve resectability in tumours located near the large hepatic and portal vessels. Reoperation is performed in cases of hepatic recurrence with a solitary metastatic lung lesion. For localized nonresectable hepatocellular carcinoma, cryosurgery, using liquid nitrogen, is promising. Of 30 cases thus treated, there were no operative deaths and the 1- and 2-year survival rates were 44.8% and 15.4% respectively. Favourable palliation of hypervascular hepatocellular carcinoma may be obtained. In 23 cases, 1- and 2-year survival rates were 27.3% and 14.3% respectively. Surgeons in Shanghai have successfully removed 132 hepatic cavernous hemangiomas, the largest being 63 × 48.5 × 40 cm and weighing 18 kg. The patient returned to active farm work. The key to their success is a new technique they developed to control the blood supply to the liver, by intermittent occlusion of all vessels in the porta hepatis, without hypothermia.

Hepatectomy is also extensively used for liver injury and intrahepatic bile-duct stones. The latter are common in China. With herbal medicine, stones may be passed, but most patients require surgery because of biliary obstruction and superimposed infection. Several types of operation have been devised to facilitate passage or removal of sandy pigment stones: left hepatic lobectomy, plastic operations for bile-duct strictures and Roux-Y anastomosis between the hepatic bile duct and jejunum. Results have been disappointing since the cause of pigment-stone formation is still unclear and recurrence frequent.

In 18 hospitals in the Shanghai and Wuhan areas, 57 orthotopic liver transplants have been performed after total hepatectomy for primary carcinoma of the liver. One patient survived 264 days, and six others longer than 6 months. Most patients died of recurrent carcinoma but one, who lived 139 days, died of extensive hemorrhage from the upper gastrointestinal tract and another, who lived 93 days, died of systemic fungal infection.

Pancreas

Pancreaticoduodenectomies for cancers of the ampulla of Vater and head of the pancreas have been done in many hospitals, and recently insulinomas have received attention. One report has described the successful removal of 60 insulinomas, all verified by pathological examination. In the diagnosis of insulinoma, the concentration of insulin and glucose in peripheral blood of the fasting patient, in portal blood intraoperatively and bioassays on portosplenic blood by percutaneous transhepatic catheterization are all emphasized. The last test usually shows a peak value at the site of the tumour.

Heart

The most outstanding work in cardiac surgery is the treatment of congenital cyanotic heart disease, including tetralogy of Fallot, Ebstein anomaly, monoventricular deformity, double right ventricular outlet accompanied by stenosis of the pulmonary artery and trilogy of Fallot.

In a series of 980 patients operated on for tetralogy of Fallot the death rate was 3.5%, late death rate 1.6%, rate of conduction block 0.1% and occurrence of residual interventricular septal defect

3%. Results compare favourably with the current literature. The operative treatment of a double right ventricular outlet accompanied by pulmonary artery stenosis was improved, and only 5 (14.3%) died in a series of 35 cases.

The Old and the New

Treatment of disease by combining traditional medicine with modern medicine is a unique feature of medical practice in China. In surgery, an outstanding example is in the management of the acute abdomen. As we know, acute abdomen consists of a number of disease entities. In Western medicine it is classified according to the pathologic condition and the organs involved. In traditional Chinese medicine, it is classified into symptom groups according to the clinical manifestations, the type of pulse and the appearance of the tongue. In the last 20 years, doctors of the two schools have studied together, learning from each other, and have gradually formed a common language in integrating both classifications. Although an acute abdomen can include disease of one or various organs, in the final analysis the pathologic process generally consists of obstruction, infection and circulatory interference. Doctors of traditional Chinese medicine treat according to the symptom groups. Evaluation by modern scientific methods has shown that some medicinal herbs used by traditional doctors for acute abdomen are beneficial in overcoming obstructions of the gastrointestinal and biliary tracts and regulating the functions of these organs: they have marked antibacterial and detoxicating effects, are effective in improving the circulation of the organs involved, in tranquillizing the central nervous system, in stimulating the activity of adrenal cortex and in enhancing general body resistance.

In general, the treatment of an acute abdomen by traditional medicine is aimed, on the one hand, directly at the causative factor and its resultant pathologic changes, and on the other, at regulation of the body's physiologic functions and enhancement of general body resistance. More than 50 000 cases of acute abdomen with presumptive diagnoses including appendicitis, perforation of peptic ulcer, cholecystitis, pancreatitis and peritonitis, have been treated by the combined method, and more than 70% of the patients recovered without surgical intervention. However, the one vital shortcoming of traditional medicine is that most herbs must be administered orally. With the presence of gastrointestinal upset, frequently seen in acute abdomen, it is at times impossible for the patient to retain a drug, especially herbs that have a disagreeable taste.

There has been considerable progress in the combined treatment of fractures. Its scope has been extended from fresh to old fractures, from closed to open and infected fractures, from shaft to intra-articular fractures, and to fractured vertebrae. Additions to methods used include "limited operations" with the alignment of fractured ends by instruments, internal bony fixation to manual reduction and intramedullary pin fixation to external short splint immobilization.

As a result of these developments, more and more types of fractures are amenable to the combined method of treatment. Another spectacular achievement in combining traditional with modern medicine is the treatment of chronic lymphedema and elephantiasis, by baking the affected limbs. In a series of 1166 cases, it was effective in 98%. Regeneration of lymph vessels has been shown by lymphangiography after such treatment.

Operations using acupuncture have been done in more than 2 million cases. In cases with good analgesic effect, the functional changes in the circulatory, gastrointestinal and neurologic systems were slight and within normal physiologic limits, whereas in those with poor analgesic effect, marked functional changes were observed. Proper choice of points for acupuncture, aimed at keeping these systems in a normal physiologic state, seems to improve the result of acupuncture. The mechanism of analgesia by acupuncture is still under extensive investigation.

Microsurgery

In the last 20 years, microsurgery has been practised at various hospitals in China. One hospital reported 45 severed distal phalanges that were reimplanted with 75.6% rate of success. One author reported on 30 severed fingers in children under 12 years of age; all were successfully reimplanted. Instead of amputation for patients with mangled wrists, the reimplantation of intact fingers to the forearm stump has been successfully performed with partial restoration of hand function in some cases. One report deals with 74 cases of transplantation of toes, together with their main blood vessels, to reconstruct thumbs or fingers, 69 (93.2%) of which were successful. Also reported is a case of successful transplantion of the second toe together with a flap of skin from the dorsum of the foot to reconstruct a thumb and cover a wound of the hand at the same time. Various musculocutaneous and other composite grafts have been devised, and anatomical studies of these flaps have been meticulous, contributing to a book entitled *Anatomy of Microsurgery*. These flaps have been used extensively in plastic and reconstructive surgery, including reconstruction of the penis.

Successful transplantation has been recorded in 40 cases, using free segments of the fibula with vascular supply to fill the gap between the ends of fractured bones and including congenital pseudoarthrosis of the tibia. Free segments of the ileum with vascular pedicles were used to connect the ends of the esophagus after esophagectomy at the cervical, thoracic and cervicothoracic levels. Five of six patients were operated upon successfully. Free skin grafting was prepared by implanting an intact portion of the greater omentum, with its vascular supply, under the skin flap as an initial stage. After circulation had been established between the omentum and the skin flap, the two were excised together as a graft and anastomosed between the gastroepiploic vessels of the omentum and the vessels of the recipient area. With this method, skin may be taken from any region, irrespective of its vascular distribution, provided it is within reach of the omentum. Microsurgical techniques have also been used for anastomosis of extra- and intracranial arteries, to treat patients with occlusive lesions of cerebral vessels. In some, anastomoses were made not only between the superficial temporal and middle cerebral arteries but also to the occipital and posterior inferior cerebellar arteries. Transplantation of a vein segment has also been done to bridge the extra- and intracranial arteries.

With microsurgery, complete extirpation of pituitary tumours can be performed without injury to the normal glandular tissue. Fifteen cases of pituitary tumour operated upon, using this technique, have been reported.

Organ transplantation is another new field of surgery in our country. Some 1000 cases of kidney transplantation have been done in Beijing, Shanghai, Wuhan and other cities. Patient survival is 50% to 70% at 1 year, 43% at 2 years and 33% at 3 years. The longest period of survival is 4.5 years and the patient is still in good condition.

Conclusions

In China, we are rather weak in research, especially in basic sciences, hence the slow and uneven progress in our surgical work. The majority of our surgical departments are not equipped with a research laboratory. We especially lack trained personnel to carry out research projects. Last, but not least, we lack up-to-date sophisticated instruments in our laboratories. As is known to all, ours is still a developing country, and a tremendous amount of work must be done to fill the gaps. We spare no efforts to learn from all developed countries, including Canada and the United States, and that is why I am here. □

PETER D. ROY, MD, FRCSC

The Value of Trauma Centres: a Methodologic Review

This review examines the rationale for the development of trauma centres in North America. The value of local and regional trauma care systems is considered, emphasizing study methodology. Evidence is acquired from case-series reports, before-and-after studies and intersystem comparisons. It overwhelmingly suggests that the main determinants of survival, given the severity of the injury, are the adequacy of the initial resuscitation and the early recognition of serious injuries. Thus, doctors involved in front-line trauma care, whether in the centre-city teaching hospital or in the rural community setting, must be properly prepared.

Cette revue regarde aux raisons pour la création et l'augmentation des centres de trauma dans l'Amérique du Nord. La valeur des systèmes local et régional est considérée avec accentuation sur la méthodologie. L'évidence est examinée des rapports de séries de cas, des études d'avant-et-d'après, et des comparisons inter-systèmes. Les preuves suggèrent profondément que les influences principales de la survie, donne la sévérité de la blessure, sont les mérites de la réanimation initiale et la reconnaissance des blessures sérieuses envers le début. Ainsi, l'intérêt essentiel dans l'éducation du trauma doit être la préparation des docteurs inclus en première ligne du soin traumatique, soit dans un centre universitaire ou dans un centre en communauté.

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In recent years there has been a resurgence of interest in the care of patients with severe traumatic injuries, particularly relating to the concept of trauma centres. Intuitively one expects the results will be improved because of the immediate availability of rapid transportation, highly trained field personnel and emergency physicians, modern diagnostic tools and experienced trauma surgeons. Who could possibly disagree? Yet, when one searches for objective evidence in support of this concept, the data are suprisingly lacking. In selected areas of North America, highly sophisticated systems of trauma care have been developed, with trained paramedical personnel, advanced communications systems, helicopter transport, well-staffed and well-stocked emergency operating rooms, special trauma fellowships and research programs.

How can we tell if all the money is well spent? More specifically, how can we objectively evaluate the performance of a trauma centre or a regional system? Ideally, we should compare the sophisticated system with another that presumably does not have such attributes. We could also compare the results of the current system with those existing before its implementation in "before-and-after" fashion. Ideally, the study should be prospective (i.e., all injured patients satisfying the entry criteria, or at least a representative sample thereof, should be studied). After stratification for age, sex, nature and severity of injury and any other predictive variables, the patient is followed up to an end-point such as death or discharge from hospital. Outcome scores could be assigned for death, disability, length of hospital stay, cost of care or whatever is desired. By examining comparative data between systems, satisfactory conclusions could then be reached using appropriate statistical techniques.

In reality, this is difficult if not impossible. The volume of serious trauma in most Canadian centres is too small for meaningful prospective comparison, particularly after allowing for stratification. Ethical considerations would preclude randomization of trauma care. The very sense of urgency discourages the surgeon from participating in clinical trials. With these considerations in mind, it is understandable that the evidence is so slim. It is of interest that Trunkey[1] has advocated a randomized, controlled trial of antishock trousers in the field to study their effectiveness.

In evaluating trauma centres, it is useful to examine statistics from nontrauma centres to see if a problem exists. This information is found in anecdotal or case-series publications.

Case-Series Reports

Van Wagoner[2] examined the hospital charts and autopsy findings of 606 American soldiers who were accidentally injured and died in hospital during the years 1957 to 1959 inclusive. The military keep good records, but the same could not be said for the hospitals where these patients died. One-sixth would have survived with the most basic prompt diagnosis and treatment, and a further one-sixth had such limited treatment that they, too, could probably have recovered with adequate care. Over one-half of the remainder died of uncomplicated head injuries; they were automatically considered unsalvageable. Even allowing for scanty records, Van Wagoner believed that this loss of life among healthy young men was appalling and that a serious problem existed.

The United States National Academy of Sciences agreed. In a classic white paper in 1966[3] it called trauma "the neglected disease of modern society". In the same year, the first trauma centre opened at the Cook County Hospital in Chicago, followed shortly by the Maryland Institute for Emergency Medical Services in Baltimore. In 1973, the US

Congress enacted funding to communities for the planning, improving or expanding of emergency medical services. Applicants were required to describe actual and potential standards of care, and to include methods of evaluating the system at regular intervals.

The case-series report, such as the one by Van Wagoner, became popular as a method of reporting and assessing the results of trauma care. A series of deaths would be studied to find out how and why the patients died. Various characteristics, such as mean injury severity score (ISS) or percentage of avoidable deaths, would be determined. This retrospective study had the effect of switching the dependent and independent variables. Death became the entry criterion, and age, sex, ISS, units of blood given, and so on became the derived results. However, this method has much to recommend it. Case finding through coroner's offices or hospital medical records is easy. Being retrospective, the study can be done in leisurely fashion. The need for statistical and epidemiologic back-up is minimal. A further refinement is added when only autopsied cases are selected for study. As described by West,[4] an ISS can be assigned, utilizing only the coroner's report, the autopsy report and the death certificate; and the death can be classified as preventable or unpreventable. The hospital chart need not be reviewed and the hospital may not know that its cases are under review.

There are problems associated with this method. First, the data may not be adequate. Without reference to the hospital chart, one cannot, for example, determine the volume of crystalloid given, how long the consultant took to arrive or how much blood was available. Moreover, the method depends on a high autopsy rate, and a cooperative coroner. However, as in some Canadian centres, it may be that autopsies are performed only on "questionable" cases, thus slanting the results toward a higher percentage of apparent errors in management.

Several series of this nature have been reported. Perry and McLellan[5] reviewed autopsy findings and coroner's reports of 127 victims of motor vehicle accidents who died in hospital in the Minneapolis area between 1958 and 1963. Of 12 patients who died of abdominal injuries alone, 1 died in the emergency department just after arrival, 1 died in the operating room of massive hemorrhage, 5 died after what appeared to be inadequate surgical technique and 5 did not have an operation that could possibly have saved them. Frey and colleagues[6] examined records of 159 motor-vehicle-accident deaths, the only autopsy cases from 450 such deaths in Washtenaw County, Michigan, during the years 1962 to 1967. Salvage was considered possible in 28 (18%). Of the 60 who reached hospital alive, the lives of 12 (20%) were thought to be potentially salvageable. Gertner and colleagues[7] examined the records of 33 patients who died in Baltimore hospitals of abdominal injuries sustained in motor vehicle accidents during the years 1964 to 1969. Seventeen (52%) were judged to have had a reasonable chance for survival, 4 were inadequately managed but would have died anyway and 12 were adequately managed. Only two (6%) of the deaths occurred in the two university hospitals, which saw 19% of all major trauma. Trunkey and Lim[8] reviewed all 425 trauma deaths in San Francisco for 1972. Of the 155 who died after arrival in hospital, 8 (5.2%) were considered to have had preventable deaths, and in another 11 (7.1%) death "might have been preventable". Only one of these deaths occurred in the San Francisco General Hospital, the only hospital with a trauma unit. Gilroy[9] reviewed the cases of 105 patients with blunt trauma in Northern Ireland in 1981 who died in hospital and for whom an autopsy was performed. Of these, 17 (16.2%) had errors in treatment that affected the outcome. The author acknowledged that improvement in the current system was possible.

Data were recently published from the Mayo Clinic[10] for the years 1976 to 1980, the first 4 years of their trauma-care program. Looking at all 111 hospital deaths after motor vehicle accidents, they found that 98 of the patients died of injuries related to the nervous system (mean ISS 49.3) and in 13 the injuries were not related to the central nervous system (mean ISS 45.6). These data compared favourably with those from other, primarily urban, centres, and the authors concluded that a rural centre (Rochester, Minn.) could also achieve good results. Similarly, Wright and colleagues[11] examined the records of 155 patients who died in the trauma unit at Sunnybrook Hospital in Toronto. Of these, autopsy was performed in 102; an ISS was assigned to each of these cases. Fifty deaths were primarily related to the central nervous system, and these cases were excluded from further study. The mean ISS for the remaining 52 cases was 48 on admission and 52 at autopsy. This was said to represent the severe nature of injury in the patients who died as a result of trauma in their institution. It was stated that "diagnostic error was not a major factor in the deaths"; management error was not discussed.

The problem with such case-series reports, of course, is that there is no denominator. We have no idea how many patients were treated well or poorly, and survived.

It is difficult, if not dangerous, to draw conclusions from a single case series. Such reviews can benefit the institution or system being reviewed, but, in the absence of comparative data, conclusions can be reached to suit the bias of the reviewer. All of the above publications were intended to argue in favour of a regional system of trauma care. However, Certo and colleagues[12] reviewed hospital deaths resulting from motor vehicle accidents in Vermont for the years 1974 to 1979. The charts of 45 patients who died of causes unrelated to the central nervous system were reviewed by a panel of surgeons. The mean ISS was 46, and 10 patients (22%) were considered to have had potentially survivable injuries. For the previous 5-year period in Vermont, Foley and associates[13] reported a potential survival rate of 26%. These data appear to be in line with other published series, although Vermont is primarily rural, with no regional system of trauma care. Of the 10 preventable deaths, in only 2 might the outcome have been altered by direct admission of the patient to a regional trauma centre. The remaining victims died of primary care errors such as failure to maintain an airway, to resuscitate adequately and to recognize a clear and pressing need for early surgical intervention. The management of these eight patients was thought to be well within the province of community general hospital care and a plea was made for better basic training in such hospitals rather than shift to the trauma-centre concept.

Injury Severity Scoring

Many scales for measuring injury severity exist. The most popular is the ISS developed by Baker and associates[14] in the early 1970s. The scale was developed as a descriptive index of trauma, and as a method of evaluating emergency care in eight Baltimore hospitals. The index is based upon specific anatomic injuries. Each injured area of the body is assigned a score on a scale of 1 (minor) to 5 (critical, survival uncertain). The three highest regional scores are then squared and added together, for a maximum possible score of 75.

There are problems with using the ISS. First, it assigns a static value to a dynamic phenomenon; for example, a tension pneumothorax (score 4) can be converted to a minor chest-wall injury (score 1) in a few moments. Second, the scale is discontinuous, certain scores are mathematically impossible to attain. Third, a single critical injury with survival uncertain (score $5^2 = 25$) appears less serious than three serious but non-life-threatening injuries (score $3^2 + 3^2 + 3^2 = 27$), when this may not be the case. Fourth, injuries to the central nervous system are more clearly related to outcome than, say, orthopedic injuries. Finally, the scale is

imprecise and the assignment of scores subjective. When scores are assigned retrospectively, in nonblinded fashion by a reviewer who wishes to prove a point, a large element of bias may be introduced.

More recently, a physiologic trauma score has been developed by Champion and colleagues[15] using respiratory rate, level of consciousness and blood pressure. Since there is less room for variation of interpretation, this scale should be more reliable.

Similar problems plague the many indices of morbidity, as, for example, the use of disability scoring. A patient can become disabled along one of three pathways: the injury may be such that disability is expected; the injury may be so severe that death is expected, but the treatment so good that the patient survived in a disabled state; or the injury may be so trivial that complete cure is expected, but treatment was so poor that disability resulted. Such data must be handled carefully in order to sort out the different sources of morbidity.

Comparisons Within a System

Patterns of outcome can be found within a single series. Holmes and Reyes[16] reviewed the charts of 41 trauma victims who were admitted to the intensive care unit at Cook County Children's Hospital. Twenty-two children were transferred from other institutions, and 19 were direct admissions. The two groups had similar mean injury severity and Glasgow coma scale scores. In the transferred group, four children died and three had long-term neurologic disability. There were no deaths and only one long-term disability in the directly admitted group. Death and disability were said to be due to delay in definitive management, and it was concluded that all children with serious traumatic injury should go directly to the regional pediatric trauma centre.

Differences within a system of trauma care can be examined. A good study[17] was done of all serious motor vehicle accidents in 1979 for six counties in and around Portland, Oregon. There was no regional trauma-care plan. Entry criteria for study purposes included admission to the intensive care unit, death in the emergency department or an operation within the first 24 hours, excluding one for an isolated extremity injury. There were 659 seriously injured patients, 135 of whom died. A trauma panel then reviewed a computerized summary of relevant data collected from all available sources, which effectively blinded them to the hospital involved. An ISS was assigned and the outcome judged in accordance with the severity of injury. The hospitals were then arbitrarily divided into small (less than 200 beds) or large. The mean ISS, time taken for arrival of the consultant, arrival of blood and for operation, if needed, were similar for the two groups. The death rate in small hospitals was 26%, compared with 17% in large hospitals, ($p < 0.01$). In small hospitals, 39 of 69 deaths occurred in the emergency department within 1 hour of admission, while in the large hospitals the number was 6 of 66 ($p < 0.0001$). This suggests poor resuscitation as a major defect leading to death in small hospitals as the late death rate was not significantly different in the two groups. The panel considered that 34 (25%) of the 135 deaths were inappropriate but did not analyse their distribution.

Another good study involving five hospitals during the years 1972 and 1973 was carried out in the Madison, Wisconsin area by Moylan and colleagues.[18] A nurse investigator surveyed all 4566 trauma charts, of which 823 met the criteria for major trauma. A review panel of university surgeons then looked at 237 of these, which included 57 deaths, 146 patients whose length of stay fell outside predetermined limits and 34 others selected at random. Overall, 16% of these patients were judged to have received unacceptable care, ranging from 6% at the University Hospital to 58% at a rural hospital. In a general-teaching hospital the care was considered unacceptable in 13%, compared with 31% and 27% at two general-community hospitals. The same authors[19] then expanded the study to be representative of the whole state. A random sample of 28 hospitals was chosen of which 8 were classified as providing area or general emergency service, 10 as community emergency service and 10 as unclassified. Random samples of trauma cases from each hospital were studied, with a heavier sampling ratio from the rural hospitals to reflect the lower case load. After eliminating cases of minor trauma, all deaths and one-third of the other cases were considered by trauma review panels. On this occasion all identification data were censored, and the panels thus blinded as to the hospital of origin. Overall, the acceptability-of-care rate for 556 cases was 70%, varying from 81% for "area and general" to 65% for "community" to 45% for "unclassified". These results were all statistically significant.

The State of Illinois spent considerable money organizing a regional system of trauma care, which was implemented in 1971. This has been the subject of numerous reports,[20,21] nearly all of the narrative variety. The only data of substance came from the Springfield area, where a before-and-after study[22] showed that the overall number of deaths from motor vehicle accidents fell by 29% after implementation of the system, while the number of injuries actually rose by 1%. This death-to-injury ratio fell 28%, from 2.5/100 to 1.8/100. This was hailed as proof of success of the program. In fact, similar data were being reported all across the United States, due mainly to the 55 mph speed limit, safer cars, introduction of the breathalyzer test and increasing use of seat belts. The overall severity of injury was simply decreasing. The Illinois literature has been the subject of criticism by Smock and Thall[23] and Willemain.[24]

The city of Jacksonville, Florida, instituted a system of emergency care[25] in the period 1968 to 1971, during which time the death-to-injury ratio fell 24%, from 1.55/100 to 1.18/100. The authors were more candid, noting that this was a national trend, and stated they had no data base to measure the overall changes in the severity of injury. However, they did note that over the same period, the death-to-injury ratio statewide in Florida fell by only 9.1%, from 1.76/100 to 1.60/100 and that in seven rural counties around Jacksonville, the ratio actually rose from 4.2 to 4.3/100. More recent state-wide data from Nebraska[26] showed an overall decline of 23.9% in the number of trauma deaths. During the period, an improved system for emergency medical services was being implemented. The authors believed that this system was responsible for much of the decline, since non-vehicular accident deaths decreased nearly as much as highway accident deaths. Safety programs in the workplace and a decrease in violent crime could also have been factors. The bulk of the decline was in pre-hospital deaths which in the authors' opinion was the result of their system of emergency medical services, but again, it could have been the result of better prevention.

Comparisons can be made within a single hospital in before-and-after fashion. Baker and colleagues[27] looked at data from Yale-New Haven Hospital before and after the implementation of a trauma service in July 1983. The overall death rate for trauma patients admitted to an intensive care unit dropped from 16.1% before to 11.7% after, and the proportion of possibly preventable deaths dropped from 32% to 15%. The numbers are too small to achieve statistical significance.

Comparisons Between Systems

There are few published data comparing the results of care in a "trauma-centre system" with those in a region having no organized trauma care. The most widely quoted study comes from San Francisco and Orange counties in California.[28] San Francisco County has one trauma centre to which all seriously injured victims are, by design, referred. At that time, Orange County had no such system, with injured patients being transported to the nearest

hospital. One hundred consecutive victims of motor vehicle accidents who died in hospital were selected for study in each region, according to the autopsy method already described.[4] After exclusion of patients transferred into one system from another county, there were 90 cases in Orange County and 92 in San Francisco available for analysis. These were divided into two groups — those related or unrelated to central-nervous-system deaths — and evaluated in nonblinded fashion by the three authors. In the unrelated group, the mean ISS was 37 in Orange County and 45 in San Francisco; of the 30 deaths in Orange County, 11 were judged to be clearly preventable and a further 11 potentially preventable; of the 16 deaths in San Francisco, only 1 was considered potentially preventable and none clearly preventable ($p < 0.0002$). Of the 60 patients who died in Orange County of injuries related to the central nervous system, 17 (28%) were considered to have lesions amenable to aggressive surgical therapy, while none of 76 deaths in San Francisco were so judged ($p < 0.0002$).

Despite the drawback of having the authors review all the cases in nonblinded fashion, when they so obviously had something to prove, the message was received quite clearly. In 1979 Orange County underwent a major overhaul of its trauma services, with the education and licensure of paramedics, and the introduction of a radio-controlled transport system to one of five designated trauma facilities. This became the subject of a before-and-after study[29] that has also been widely quoted. A study period of 12 months in the years 1977 and 1978 was chosen. The charts of 183 patients who died in hospital were eligible for the study. This number was reduced to 100 by a process called "stratified randomization". By this method, all deaths from smaller hospitals were included, and the remainder were drawn at random from the large hospitals to make up 100 cases. These deaths were reviewed by a panel that studied the hospital records as well as the coroner's reports. Prehospital deaths, burns, motor vehicle accidents outside Orange County and late deaths unrelated to the management of the initial injury were then excluded, reducing the deaths studied to 58. The panel was aware of the hospital concerned. The "before" study was actually done before implementation of the regional system. The panel judged "salvageability" in each case, given optimal care conditions. A similar study, using identical methodology, was done to assess deaths in 1980 and 1981. The sample size of the "after" study was 60. In the "before" group, 34% (20 of 58) of victims were judged to be potentially salvageable, while 15% (9 of 60) in the "after" group were so judged ($p < 0.02$). For deaths unrelated to the central nervous system, the corresponding figures were 86% (18 of 21) and 40% (8 of 20) ($p < 0.001$). In the "after" group, 47 deaths occurred in designated trauma facilities, and the potential for survival was present in only two cases. Thirteen patients, mistakenly taken to nondesignated hospitals, died; of these 7 were potential survivors ($p < 0.0002$). The mean ISS rose from 42.5 "before" to 52 "after". Despite obvious flaws in the study design, including the built-in bias of "stratified randomization", and the nonblinding of panel members, the results were striking.

A study was recently reported from Sunnybrook Hospital in Toronto,[30] using the previously unpublished data[11] on 52 patients who died in their trauma centre, who had autopsy and who did not die of head injury. The authors then studied a sample of 202 autopsy records from the coroner's office of Ontario, trauma victims who died in hospitals other than Sunnybrook during the same period. After excluding deaths related to the central nervous system, the 103 remaining patients were assigned a postmortem ISS by the same reviewer who had studied the deaths from Sunnybrook. No "error analysis" was performed. The final result was that the mean ISS from Sunnybrook was 53, while that from other institutions was only 33. Since patients dying in the trauma facility were more seriously injured, they must perforce have received superior care. Such an analysis is seriously flawed. As mentioned before, a postmortem ISS is an inappropriate outcome variable. Only selected patients are taken directly to Sunnybrook, most of their trauma admissions being transfers from other institutions. This means that the transfers to Sunnybrook are selected from the early survivors, while the early nonsurvivors die in their home hospital. Bias inherent in the "autopsy method" has been referred to previously. The method of sampling from the coroner's office is not discussed. There is no distinction, for example, between university and nonuniversity hospitals. In southern Ontario, university hospitals receiving trauma victims are *de facto* regional trauma centres. Finally, as seen in the California report,[28] an unblinded "us versus them" study is clearly subject to bias.

Trauma Registries

Ideally, a trauma registry can be used to describe the epidemiology of trauma within a given population, and risk factors can be defined for accident occurrence, morbidity and mortality. The registry can be used as a guide for the deployment of health services and may act as a monitor of the efficacy of new health-care delivery systems, such as paramedics or helicopters. The registry may act as an indirect measure of the cost of health care related to trauma.

The history of trauma registries has not been especially bright. This is mainly because good data must be collected in prospective and goal-directed fashion. Retrospective data tends to suffer from a common problem — the generally dismal state of hospital medical records. Often there is no indication on the hospital chart of the time or nature of the accident, or whether the victim was drinking or wearing a seat belt. Furthermore, ICD-9-CM coding[31] by medical records personnel is often incomplete in multiple-injury cases.

All registries are plagued by a common problem, the inability to generalize the data. When the registry is based on data from one hospital alone, the data will reflect the referral pattern of that centre. For example, in Toronto, the Sunnybrook Trauma Unit has a very high

Trauma Mortality Reviews — Hospital Deaths

Series	No. of deaths	No. (%) of deaths possibly preventable	Type of trauma
Van Wagoner, 1961[2]	606	200 (33)	All
Perry and McLellan, 1964[5]	12	10 (83)	MVA-abdominal
Frey and associates, 1969[6]	60	12 (20)	MVA-all
Gertner and associates 1972[7]	33	17 (52)	MVA-abdominal
Trunkey and Lim, 1974[8]	155	19 (12)	All
Foley and associates, 1977[13]	43	11 (26)	MVA-abdominal
West and associates. 1979[28]			MVA-all
Orange County	90	39 (43)	
San Francisco County	92	1 (1)	
Certo and associates, 1983[12]	45	10 (22)	MVA-non-CNS
Lowe and associates, 1983[17]	135	34 (25)	MVA-all
Gilroy, 1984[9]	105	17 (16)	All blunt
Cales, 1984[29]			MVA-all
Before	58	20 (34)	
After	60	9 (15)	
Baker and associates, 1985[27]			All
Before	31	10 (32)	
After	20	3 (15)	

MVA = motor vehicle accident, CNS = central nervous system.

proportion of patients referred from other hospitals, often with severe head or spinal injury and frequently arriving by helicopter ambulance. On the other hand, St. Michael's Hospital in downtown Toronto handles a much higher proportion of off-the-street trauma, probably reflecting a more acutely injured type of patient. The number of seriously injured patients might be the same, but the patterns of injury, and hence the outcome, will be different. On the other hand, if the registry is population-based or area-based, serious problems can arise from underreporting. The data base becomes slanted in favour of interesting or serious cases.

A glaring example of this occurred in the Illinois experience referred to earlier.[22] As part of this ambitious project, a complex, computerized, online registry was established, whereby all trauma admissions from 33 designated trauma centres would be entered into the registry by specially trained personnel.[32,33] They attempted to collect everything; over 700 fields of data could potentially be noted on a single record. Needless to say, it was extremely difficult to get reports registered completely, and many cases were not reported at all. The entire system folded for lack of federal funds in 1976. An independent post mortem of the registry[34] involved random sampling of 10% of trauma patient charts from the 33 involved hospitals. Only 36.9% of these cases appeared in the registry. For the sampled cases appearing in the registry, the death rate was 5.0%, and for those not appearing in the registry, it was 1.5%. Similarly, reported patients were more frequently treated in the intensive care unit and were more seriously ill as a group. Clearly, the usefulness of the registry as a research tool was severely limited by the inability to generalize the data.

Recently, Cales and colleagues[35] described the development of a microcomputerized regional trauma registry, using comprehensive data input from the medical records of trauma hospitals and abbreviated input from nontrauma hospitals and coroner's office data. Data collection is prospective and is done with the help of coordinators who are responsible for abstracting and entering data.

The only other area-based registry report comes from Manitoba.[36] This is based entirely upon data culled from the Manitoba Health Services Commission ICD-9-CM[31] accidental injury tape and from provincial vital statistics fatality data for the years 1980 to 1982. A computer algorithm assigned each coded injury an injury severity score, and death rates were calculated by severity of injury and level of hospital providing the initial care. The data provided give useful "injury counts" and provide interesting comparisons between hospital levels, but the results are subject to the restrictions of retrospective analysis, namely, incompleteness of charting and coding by medical records. The authors did, however, conduct a validation survey of charts and found the completeness and accuracy of diagnosis to be over 95%.

Hospital-based registries have been reported from San Diego,[37] Baltimore[38] and Wheeling, West Virginia.[39] These reports deal with the methodology of setting up the registry but do not actually give results of any of the data collected so far.

The American College of Surgeons is currently in the midst of a multicentre study of trauma, which is basically a registry. Data are being collected regarding risk factors, physiologic and anatomic trauma scores and outcome. The Trauma Association of Canada, through its Registry and Research Committee, is actively trying to organize a similar study, since the nature of trauma in Canada is very different from that in the US.

Conclusions

With respect to the method of evaluation of trauma centres, several conclusions can be reached. First, retrospective studies that only look at deaths are no longer good enough. They provide useful information for internal clinical appraisal of the institution involved, but they are subject to bias. Also, randomized clinical trials are impractical, due to small numbers, the heterogeneous nature of the disease and problems related to ethics and compliance. The most practical method of evaluation lies somewhere in between. Data must be collected prospectively, which mandates the formation of some form of trauma registry. Entry criteria based on type and severity of injury must be rigorously and universally applied, which means that all entries must be studied. Allowances must be made for patients being transferred into, out of, or within the system under study. Only then can objective outcome measurements be made and cases be sampled for quality-of-care review. The development of sophisticated microcomputer hardware and software has made the technical components of data collection easier, but there is still a heavy requirement in terms of manpower and dedication in order to keep the information up to date.

In several of the mortality reviews,[2-7,12,13,17] the authors concluded that many of the "preventable" deaths occurred because of basic errors in the initial phase of treatment. Surely, one of the main reasons that trauma centres save lives is the generally excellent level of resuscitation and initial evaluation in their receiving areas? But this is no excuse for poor initial care at the periphery. The impact of trauma centres on mortality and morbidity is difficult to measure, but, with the provision of competent resuscitation and general surgical treatment in peripheral hospitals, the need for on-the-scene paramedics, helicopter transport or a "prima donna" trauma surgeon are surely reduced.

In large cities the designation of trauma centres certainly makes economic and administrative sense, but in Canada a great deal of trauma occurs away from urban centres. I would suggest that the best value for money is to teach emergency physicians the basics of resuscitation and evaluation through such programs as the Advanced Trauma Life Support Course,[40] and from ongoing education of community general surgeons who know when and how to operate.

References

1. TRUNKEY DD: Is ALS necessary for pre-hospital trauma care? (E) *J Trauma* 1984; 24: 86–87
2. VAN WAGONER FH: A three-year study of deaths following trauma. *J Trauma* 1961; 1: 401–408
3. *Accidental Death and Disability: the Neglected Disease of Modern Society*, US National Academy of Sciences, National Research Council, Washington, DC, 1966
4. WEST JG: An autopsy method for evaluating trauma care. *J Trauma* 1981; 21: 32–34
5. PERRY JF JR, MCLELLAN RJ: Autopsy findings in 127 patients following fatal traffic accidents. *Surg Gynecol Obstet* 1964; 119: 586–590
6. FREY CF, HUELKE DF, GIKAS PW: Resuscitation and survival in motor vehicle accidents. *J Trauma* 1969; 9: 292–310
7. GERTNER HR JR, BAKER SP, RUTHERFORD RB, et al: Evaluation of the management of vehicular fatalities secondary to abdominal injury. *J Trauma* 1972; 12: 425–431
8. TRUNKEY DD, LIM RC JR: Analysis of 425 consecutive trauma fatalities: an autopsy study. *JACEP* 1974; 3: 368–371
9. GILROY D: Deaths from blunt trauma: a review of 105 cases. *Injury* 1984; 15: 304–308
10. MUCHA P JR, FARNELL MB, CZECH JM, et al: A rural regional trauma center. *J Trauma* 1983; 23: 337–340
11. WRIGHT CS, MCMURTRY RY, HOYLE M, et al: Preventable deaths in multiple trauma: review of deaths at Sunnybrook Medical Centre Trauma Unit. *Can J Surg* 1983; 26: 20–23
12. CERTO TF, ROGERS FB, PILCHER DB: Review of care of fatally injured patients in a rural state: 5-year followup. *J Trauma* 1983; 23: 559–565
13. FOLEY RW, HARRIS LS, PILCHER DB: Abdominal injuries in automobile accidents: review of care of fatally injured patients. *J Trauma* 1977; 17: 611–615
14. BAKER SP, O'NEILL B, HADDON W JR, et al: The injury severity score: a method for describing patients with multiple injuries and evaluating emergency care. *J Trauma* 1974; 14: 187–196
15. CHAMPION HR, SACCO WJ, CARNAZZO AJ, et al: Trauma score. *Crit Care Med* 1981; 9: 672–676
16. HOLMES MJ, REYES HM: A critical review of urban pediatric trauma. *J Trauma* 1984; 24: 253–255
17. LOWE DK, GATELY HL, GOSS JR, et al: Patterns of death, complication, and error in the management of motor vehicle accident victims: implications for a regional system of trauma care. *J Trauma* 1983; 23: 503–509
18. MOYLAN JA, DETMER DE, ROSE J, et al: Evaluation of the quality of hospital care for major trauma. *J Trauma* 1976; 16: 517–523
19. DETMER DE, MOYLAN JA, ROSE J, et al: Regional categorization and quality of care in major trauma. *J Trauma* 1977; 17; 592–599
20. BOYD DR: A symposium on The Illinois Trauma Program: a systems approach to the care of the critically injured. Introduction: a controlled systems approach to trauma patient care. *J Trauma* 1973; 13: 275–276
21. Idem: Efforts to improve emergency medical services: the Illinois experience. *JACEP* 1977; 6: 209–217
22. BOYD DR, PIZZANO WA, ROMANO TL, et al: Regionalization of trauma care: the Illinois experience. In NYHUS LM (ed): *Surgery Annual*, ACC, New York, 1969: 25–52
23. SMOCK SN, THALL ML: Negative impacts of the Model EMS System: it is time for an honest reappraisal. *JACEP* 1977; 6: 206–208

24. WILLEMAIN TR: The status of performance measures for emergency medical services. *JACEP* 1975; 4: 143–151
25. WATERS JM JR, WELLS CH: The effects of a modern emergency medical care system in reducing automobile crash deaths. *J Trauma* 1973; 13: 645–647
26. ORNATO JP, CRAREN EJ, NELSON NM, et al: Impact of improved emergency medical services and emergency trauma care on the reduction in mortality from trauma. *J Trauma* 1985; 25: 575–579
27. BAKER CC, DEGUTIS LC, DESANTIS J, et al: Impact of a trauma service on trauma care in a university hospital. *Am J Surg* 1985; 149: 453–458
28. WEST JG, TRUNKEY DD, LIM RC: Systems of trauma care. A study of two counties. *Arch Surg* 1979; 114: 455–460
29. CALES RH: Trauma mortality in Orange County: the effect of implementation of a regional trauma system. *Ann Emerg Med* 1984; 13: 1–10
30. WRIGHT CS, MCMURTRY RY, PICKARD J: A postmortem review of trauma mortalities — a comparative study. *J Trauma* 1984; 24: 67–68
31. *International Classification of Diseases, 9th Rev, Clinical Modification,* United States National Center for Health Statistics, Ann Arbor, Mich, 1978
32. BOYD DR, LOWE RJ, BAKER RJ, et al: Trauma registry. New computer method for multifactorial evaluation of a major health problem. *JAMA* 1973; 223: 422–428
33. BOYD DR, LOWE RJ, SHEAFF LC, et al: A profile of the trauma registry. *J Trauma* 1973; 13: 316–320
34. GOLDBERG J, GELFAND HM, LEVY PS, et al: An evaluation of the Illinois trauma registry: the completeness of case reporting. *Med Care* 1980; 18: 520–531
35. CALES RH, BIETZ DS, HEILIG RW JR: The trauma registry: a method for providing regional system audit using the microcomputer. *J Trauma* 1985; 25: 181–186
36. BURNS CM: Overview of the ICD (CM-AIS-ISS) provincial trauma registry (abstr). *J Trauma* 1984; 24: 649
37. CHARTERS AC, BAILEY JA: Experience with a simplified trauma registry: profile of trauma at a university hospital. *J Trauma* 1979; 19: 13–17
38. HALLER JA JR, SIGNER RD, GOLLADAY ES, et al: Use of a trauma registry in the management of children with life-threatening injuries. *J Pediatr Surg* 1976; 11: 381–390
39. PHILLIPS LP, ZEIK AJ: Development of a computerized trauma registry. *Emerg Med Serv* 1982; 11: 14, 16
40. *Advanced Trauma Life Support Course Manual,* American College of Surgeons, Chicago, 1984

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Peripheral Nerve Injuries During Carotid Endarterectomy

Peripheral nerve injuries associated with carotid endarterectomy are fairly common but not emphasized in reported results of carotid endarterectomy. The sensory nerves to the submandibular skin and ear lobe are often damaged. Motor nerves VII, IX, X, XI and XII may be injured at surgery. The commonest motor injuries involve the facial, vagus and hypoglossal nerves. Carotid endarterectomy was studied prospectively over 1 year to document the incidence of nerve injury. Nerve injury occurred in 12% of patients, with facial and vagus nerves being involved in 4% each. Careful surgical technique based on appropriate anatomical knowledge can prevent most of these complications.

Les lésions des nerfs périphériques qui surviennent en cours d'endartériectomie carotidienne sont assez fréquentes. Elles ne sont toutefois pas soulignées dans les résultats publiés sur l'endartériectomie carotidienne. Les nerfs sensitifs vers la peau sous-maxillaire et le lobe de l'oreille sont souvent endommagés. Les nerfs moteurs VII, IX, X, XI et XII peuvent être touchés en cours de chirurgie. Les atteintes motrices les plus fréquentes concernent les nerfs facial, vague et hypoglosse. Dans le but d'évaluer la fréquence des lésions nerveuses, on a étudié prospectivement l'endartériectomie carotidienne au cours d'une période d'un an. Des lésions nerveuses sont survenues chez 12% des patients, avec une atteinte des nerfs facial et vague dans 4% des cas chacun. Une technique chirurgicale minutieuse s'appuyant sur des connaissances anatomiques appropriées peut prévenir la plupart de ces complications.

Since the first successful carotid reconstruction described by Eastcott and colleagues[1] in 1954, carotid endarterectomy for occlusive disease has become one of the most frequently performed operations in the United States.

Although neurologic deficit due to cerebral ischemia during or after the operation is the complication attracting most attention, peripheral nerve injury as a result of intraoperative trauma is a commoner complication and one that can give rise to serious postoperative sequelae. Injuries to the cutaneous nerves are usually of minor consequence. Facial nerve injury leads to cosmetic defects or minor functional impairment when only the ramus mandibularis is affected. Vagal and hypoglossal injuries, however, can cause serious postoperative respiratory complications and difficulty in swallowing.

Most of these complications are transient and therefore not serious. Some nerve injuries are asymptomatic and may be undiagnosed unless a careful postoperative examination is performed. Hoarseness has frequently been attributed to local trauma by the endotracheal tube. This study reports our findings in a consecutive series of patients operated upon by two of us (A.R.D. and C.R.L.).

Patients and Methods

All patients who underwent carotid endarterectomy at the Health Sciences Centre in Winnipeg between July 1982 and June 30, 1983 were included in the study. During this period 96 carotid endarterectomies were performed on 86 patients. The indications for the procedure are shown in Table I. All patients had undergone selective arch and carotid angiography preoperatively usually with access through a femoral artery. There were no direct carotid punctures and no neurologic deficits were observed after angiography. Most patients were examined preoperatively and postoperatively by a member of the otolaryngology service as well as by the vascular staff members. Particular attention was paid to examination of cranial nerves VII, IX, X and XII and the cervical sensory nerves. Indirect laryngoscopy was used to assess the vagus nerve. When abnormalities were discovered postoperatively, patients were followed up for as long as 18 months to observe the degree of recovery.

The usual sensory deficit in the submandibular skin due to transection of the transverse cervical nerve was not recorded. The sensation on the ear lobe in the distribution of the greater auricular nerve was usually documented.

Table I—Carotid Endarterectomy Indications for Operation (N = 96)

Indication	No. of procedures
Asymptomatic stenosis	14
Amaurosis fugax	27
Transient ischemic attack	18
Transient ischemic attack and previous cerebrovascular accident	25
Previous cerebrovascular accident	12

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Operative Technique

A modified oblique incision is made below the mastoid process to midway between the thyroid cartilage and the suprasternal notch. The greater auricular nerve is usually identified and dissected free to allow the wound edges to be retracted with a self-retaining retractor. The platysma muscle is divided and the dissection is carried down to the second plane, reflecting the sternomastoid muscle. The common facial vein is divided between suture-ligatures and the common carotid artery is then exposed within the sheath. The common carotid is carefully encircled with a tape to avoid the vagus nerve. The superior thyroid artery is then dissected close to its origin and a 2-0 silk loop passed around it. The external carotid artery and its branches are identified and a vessel loop is passed around its origin. The hypoglossal nerve is identified and its relation to the carotid bifurcation is ascertained. It is often necessary to divide the descendens hypoglossi to allow retraction of the nerve. The artery to the sternomastoid muscle, originating from the occipital artery, should be divided. Similarly, several small veins cross the nerve at this site. Their careful identification and division between small clips will greatly facilitate atraumatic mobilization of the hypoglossal nerve as described by Imparato and associates.[2] Occasionally, for a high exposure of the internal carotid artery, it is helpful to divide the occipital artery as it crosses the hypoglossal nerve. The internal carotid artery is mobilized distal to gross disease and finally the carotid bulb is completely dissected. It is important to dissect close to the artery to avoid the vagus nerve between the jugular vein and carotid artery. Rarely, the vagus nerve lies anterior or medial to the internal carotid artery, in which case it must be carefully dissected and preserved. No attempt is made to visualize the superior laryngeal nerve. The carotid sinus nerve is not usually divided.

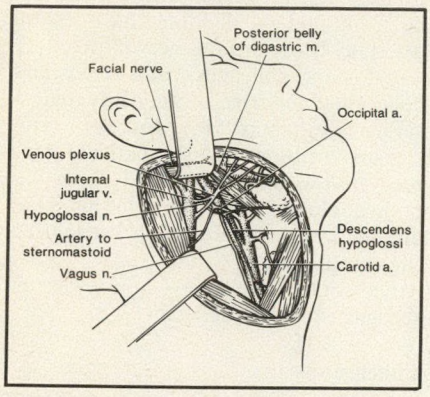

FIG. 1—Exposure of carotid artery illustrating nerves at risk and potential injury to facial nerve with upper retractor.

Table II—Peripheral Nerve Injuries After Carotid Endarterectomy

Nerve involved	No. of injuries
Greater auricular	4
Facial	
Ramus mandibularis	3
Complete	1
Vagus	4

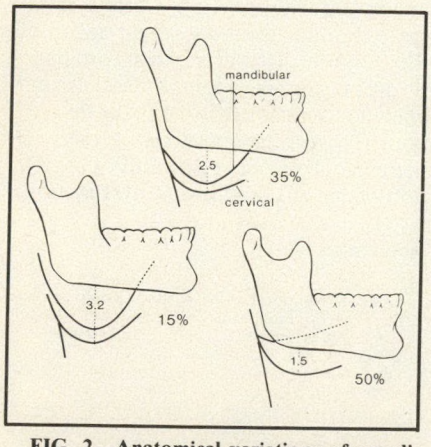

FIG. 2—Anatomical variations of mandibular and cervical branches of facial nerve (reproduced by permission from SKANDALAKIS JE JR: *Anatomical Complications in General Surgery*, McGraw, New York, 1983: 5).

Table III—Vagal Nerve Injuries: Local Complications Accompanying Hoarseness

Complication	No. of complications
Laryngeal edema	5
Hematoma, false cords	2
Tracheitis, pharyngitis	3
Vocal cord paresis	4

Results

Of the 96 cases examined, 12 were complicated by peripheral nerve deficits (Table II).

Four patients suffered greater auricular nerve palsies, manifested as a sensory loss in the region of the ear lobe. In each case the deficit was still present when the patient was discharged from hospital but caused little discomfort.

Four patients sustained a facial nerve injury. In three the injury involved the ramus mandibularis, arising from the mandibular or cervical branch of the facial nerve (Fig. 2). This results in a cosmetic defect so that the angle of the lip cannot be depressed, but no incompetence is produced. One patient sustained a transient motor deficit, involving all branches of the facial nerve. It was probably due to retraction of the posterior belly of the digastric muscle (Fig. 1) and had resolved completely within 2 weeks.

By indirect laryngoscopy four other patients were found to have decreased or absent unilateral vocal cord movement postoperatively. In all cases, this was accompanied by hoarseness. One patient had a paralysed vocal cord on the ipsilateral side preoperatively. Postoperative examination of the oropharynx and larynx revealed several local complications that were often accompanied by hoarseness (Table III). No hypoglossal nerve injuries were documented in this series although we had seen this complication previously.

Follow-up in 11 of 12 patients with postoperative complications ranged from 12 to 18 months.

At the time of discharge, 3 or 4 days after operation, the greater auricular nerve deficit was still present, but it diminished over the months. The facial nerve palsy resolved within 2 weeks and one ramus mandibularis nerve deficit remained at completion of the study. One cord paresis resolved before discharge from hospital and the remaining three within 6 months.

Discussion

The 12% rate of peripheral nerve injuries during carotid endarterectomy in our series is in agreement with that reported by Astor and colleagues[3] and Matsumoto and associates,[4] but is lower than those reported by Dehn and Taylor[5] and Evans and colleagues.[6] These differences may be related to the methods used in evaluating nerve injuries.

Greater Auricular Nerve

Most authors exclude sensory examination in the postoperative assessment of patients, since many such losses are transient and of little consequence to the patient. However, Dehn and Taylor[5] reported a very high incidence of cutaneous nerve injury with persistence of symptoms in a number of patients. Our recorded rate of injury to the greater auricular nerve (4%) may be an underestimate, but we do make a conscious effort to preserve this nerve. This can usually be accomplished by dissecting the nerve with the upper flap to allow retraction without division. The transverse cervical nerve is usually divided causing

numbness in the submandibular region and can be annoying to the man who shaves with a safety razor.

Facial Nerve

The most common facial nerve deficit following carotid surgery is paresis of the depressor anguli oris and depressor labii oris. This is usually attributed to the ramus mandibularis branch of the facial nerve but may be due to injury to the more susceptible cervical branch.[4] This injury may be permanent, as in one patient in our series. The defect caused labial asymmetry but not drooling or labial incompetence as has been suggested.[5] There were four facial nerve injuries for an incidence of 4% which is comparable to that reported by others.[3-5,7]

The marked variation in the anatomy of the mandibular and cervical branches of the facial nerve makes them susceptible to injury in a high neck incision (Fig. 2). It has been stressed that the upper end of the incision should be placed well posterior to the ramus mandibularis, in line with the mastoid process. In most instances the lesion is a neuropraxia, probably due to the use of a self-retaining retractor. One patient had a total facial nerve palsy in our series; fortunately it resolved within 2 weeks. This injury was probably due to a retractor causing direct pressure on the trunk of the facial nerve. This can best be avoided by using the oblique incision and high exposure of the internal carotid artery without the need for a retractor.

Vagus Nerve

The most serious nerve injury receiving attention in the past few years is that to the vagus nerve, with resulting vocal cord paresis and motor speech dysfunction. This complication can cause serious postoperative respiratory distress and difficulty in swallowing. The principal branches from the vagus nerve are the superior laryngeal and the recurrent laryngeal nerves (Fig. 3). The superior laryngeal divides into internal and external laryngeal branches. The external branch is motor to the inferior constrictor and the cricothyroid which tenses the vocal cord and is responsible for pitches in the upper range. The internal branch is sensory to the larynx and is protective to the airway. We evaluated the vagus nerve by preoperative and postoperative indirect laryngoscopy and found cord paresis in four patients. Using similar techniques of examination, Astor and colleagues[3] and Matsumoto and associates[4] had a similar incidence. However, Dehn and Taylor[5] found a vocal cord palsy in 25.5% of their patients and Evans and associates[6] found vagal nerve dysfunction in 35% by using a speech pathologist's assessment of function. Astor and colleagues[3] identified the superior laryngeal nerve injury separate from the recurrent nerve.

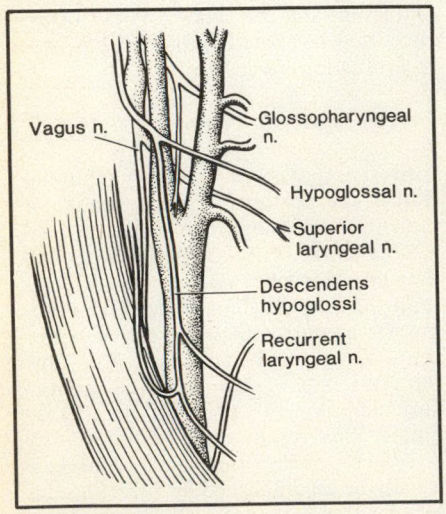

FIG. 3—Relationships of cranial nerves to carotid artery bifurcation.

It is apparent that the recorded incidence of vagal nerve dysfunction is variable and is related to the methods of assessment postoperatively. The majority of vagal nerve injuries resolve, although it may take 6 months or longer, but the injury may be permanent.[3,6]

Preoperative assessment is essential, particularly if the patient has had previous neck surgery such as carotid or thyroid operations. It is important to recognize that recurrent nerve palsy may be asymptomatic.[3] During exposure of the common and internal carotid arteries the vagus nerve should be visualized and the arteries dissected free from the nerve. Care must be taken to avoid the nerve when applying the vascular clamps. The superior laryngeal nerve passes posterior to the external carotid artery and is in close proximity to the superior thyroid artery (Fig. 3). The arteries should be dissected free before encircling with loops so that the nerve is free when the vascular clamps are applied. It must be recognized that the vagus nerve may sometimes be anterior to the carotid artery.

Hypoglossal Nerve

Injury to the hypoglossal nerve has been the one most commonly recognized in carotid endarterectomy and is at greatest risk because of its proximity to the carotid bifurcation.[4,8] Unilateral nerve palsy will cause some difficulty in swallowing and articulation. Bilateral hypoglossal nerve palsy can cause upper airway obstruction as reported by Bageant and colleagues.[9] Imparato and associates[2] have emphasized the importance of mobilizing the hypoglossal nerve to reduce the incidence of postoperative paresis of the tongue. We use careful division of the venous plexus over the hypoglossal nerve and division of the sternomastoid artery.

Division of the descendens hypoglossi will also allow better exposure of the internal carotid artery without direct retraction on the nerve. More distal exposure of the internal carotid artery can be facilitated by dividing the occipital artery as it crosses the nerve.

Astor and colleagues[3] and Evans and colleagues[6] reported a rate of hypoglossal nerve injury of 5%. Most such injuries are transient and the patients have recovered within a few days.[7] There was no hypoglossal nerve injury in this series although we have previously recognized the complication.

Glossopharyngeal nerve injury was not recognized in our series.

Conclusions

Peripheral nerve injuries are common complications of carotid endarterectomy. Injury to the vagus and hypoglossal nerves can have very serious postoperative sequelae. The frequency of these nerve injuries is higher when functional assessment is used. All patients who have had previous head and neck surgery are at risk of having an asymptomatic cord paresis or tongue dysfunction. All patients who undergo carotid endarterectomy should have a preoperative and postoperative evaluation of vocal cord and swallowing function. Careful surgical technique should prevent most of these injuries. However, when cord paresis does occur and a contralateral carotid operation is planned, it should be delayed until recovery has occurred. Otherwise there may be severe respiratory obstruction or serious swallowing dysfunction.

References

1. EASTCOTT HG, PICKERING GW, ROB CG: Reconstruction of internal carotid artery in a patient with intermittent attacks of hemiplegia. *Lancet* 1954; 2: 994–996
2. IMPARATO AM, BRACCO A, KIM GE, et al: The hypoglossal nerve in carotid arterial reconstructions. *Stroke* 1972; 3: 576–578
3. ASTOR FC, SANTILLI P, TUCKER HM: Incidence of cranial nerve dysfunction following carotid endarterectomy. *Head Neck Surg* 1983; 6: 660–663
4. MATSUMOTO GH, COSSMAN D, CALLOW AD: Hazards and safeguards during carotid endarterectomy. Technical considerations. *Am J Surg* 1977; 133: 458–462
5. DEHN TC, TAYLOR GW: Cranial and cervical nerve damage associated with carotid endarterectomy. *Br J Surg* 1983; 70: 365–368
6. EVANS WE, MENDELOWITZ DS, LIAPIS C, et al: Motor speech deficit following carotid endarterectomy. *Ann Surg* 1982; 196: 461–464
7. HERTZER NR, FELDMAN BJ, BEVEN EG, et al: A prospective study of the incidence of injury to the cranial nerves during carotid endarterectomy. *Surg Gynecol Obstet* 1980; 151: 781–784
8. DEWEESE JA, ROB CG, SATRAN R, et al: Results of carotid endarterectomies for transient ischemic attacks — five years later. *Ann Surg* 1973; 178: 258–264
9. BAGEANT TE, TONDINI D, LYSONS D: Bilateral hypoglossal-nerve palsy following a second carotid endarterectomy. *Anesthesiology* 1975; 43: 595–596

HUGH U. CAMERON, MB, CH B, FRCSC*

Role of Total Knee Replacement in Failed Knee Fusions

The author describes his technique and the results of total knee replacement in four patients who already had fused knees. In two, the patella was present so a semiconstrained prosthesis was used, but in the other two a stabilized knee prosthesis was used because the patella was absent. In the first case, lack of experience with the technique led to failure of the prosthesis and revision was necessary, but in subsequent cases the patients have done well.

Quadriceps control was not a problem. Range of movement improved slowly over the first year. The author concludes that disassembly of knee fusion is possible and gives acceptable results.

L'auteur décrit sa technique et les résultats obtenus de pose des prothèses totales du genou chez quatre patients qui avaient déjà une fusion des genoux. Dans deux cas, la rotule était présente, de sorte qu'une prothèse semi-contrainte fut utilisée. Dans les deux autres cas, la rotule étant absente, on opta pour une prothèse du genou stabilisée. Dans le premier cas, le manque d'expérience avec la technique entraîna un échec et la prothèse dut être révisée; dans les cas subséquents, les résultats furent bons.

Le contrôle des quadriceps n'a pas posé de problème. L'amplitude des mouvements s'est améliorée lentement au cours de la première année. L'auteur conclut que la séparation d'un genou fusionné est possible et que l'opération donne des résultats acceptables.

Although disassembly of the fused hip is now standard procedure, nothing has yet appeared in the literature on disassembly

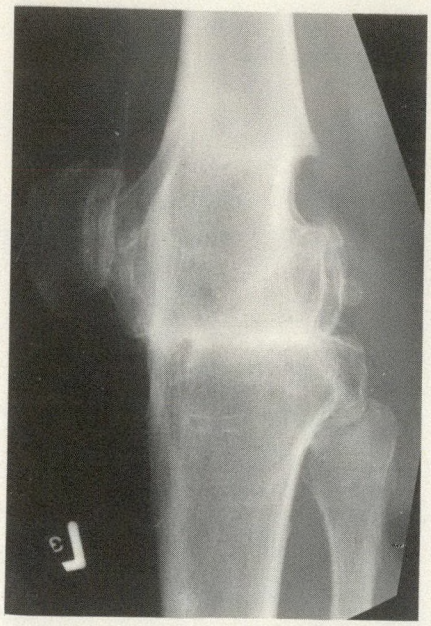

Fig. 1a

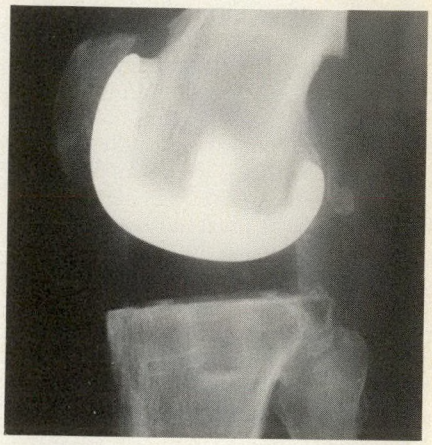

Fig. 1c

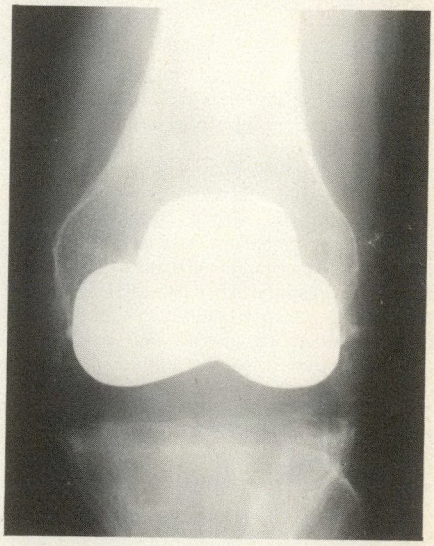

Fig. 1d

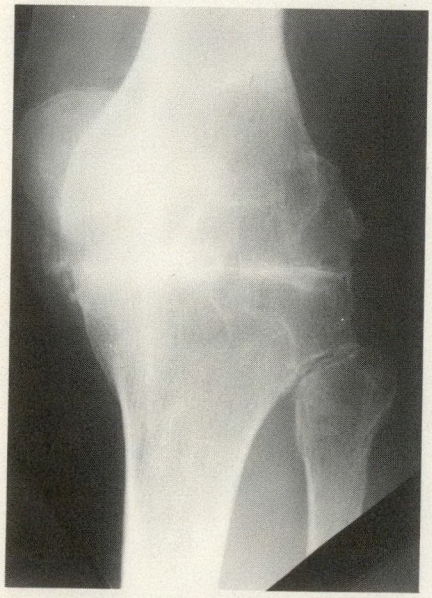

Fig. 1b

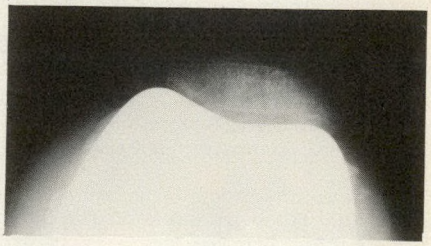

Fig. 1e

FIG. 1—Case 1. (a and b) Knee that was fused 18 years before admission. (c and d) Fusion was disassembled and semiconstrained total knee replacement performed. Uncemented tibial component held in place by ridged plastic pegs was used with cemented femoral component. (e) Patelloplasty only was performed.

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of the fused knee. Knee fusion creates such a grave disability that no patient is truly happy with it.

Over the last 7 years I have done four knee fusion disassemblies. This report examines the technique and results. Those with a fibrous ankylosis and a few degrees of motion were excluded from the series.

Technique

The joint is entered through a medial parapatellar incision, usually the site of a previous incision. If fused to the femoral condyles, the patella is mobilized using an osteotome and can then be dislocated laterally. A periosteal elevator is used to elevate the capsule off the femur and proximal 1 cm of tibia, both medially and laterally, around to the back of the knee. A 2-cm wedge of bone is resected, leaving the posterior cortex intact. When the posterior cortex can be clearly visualized it is gently multiply cracked with a ½-inch osteotome. The knee is then gently flexed to fracture the posterior cortex and the capsule is elevated from the posterior part of the femoral condyles and tibia. Knee replacement can then proceed as usual.

Case Reports

Case 1

This 81-year-old woman had undergone knee fusion 50 years previously for septic arthritis. Severe osteoarthritis developed in the other knee. A knee replacement was performed on the nonfused side. She was so pleased with the result that she asked for the fusion of the other knee to be disassembled and a replacement done. This was accomplished and an I.C.L.H. (Protek, Switzerland) knee prosthesis inserted. The patella was present. Unfortunately the tibial component was inserted with the tibia too externally rotated so the patient failed to achieve adequate movement. In ignorance of the error, a manipulation was carried out under anesthesia and resulted in disruption of the medial collateral ligament. The resulting instability necessitated revision to a Guepar procedure 9 months later.[1] Seven years later the knee is stable and pain free with a range of movement of 0° to 100°.

Case 2

A 60-year-old man had undergone a high tibial osteotomy. He failed to regain movement and subsequently, during manipulation under anesthesia, he suffered a supracondylar fracture. The knee was therefore fused (Fig. 1a and b).

When seen 15 years later he had severe osteoarthritis in the opposite knee. The knee fusion was disassembled and a Tricon P (Richards Manufacturing Co., Memphis, Tenn.) prosthesis inserted. At 3½ months the range of movement in the knee was from 0° to 35° and this slowly improved over the next year to a range of from 0° to 90°. The knee is stable and pain free. Subsequently this man's other knee was replaced.

Case 3

A 65-year-old woman had had her knee fused for osteoarthritis. A patellectomy was carried out at the same time. Eighteen months later the fusion was disassembled and a Guepar hinge inserted. In 6 months the range of movement had improved rapidly to 90° and 4 years later, it was from 0° to 110°. She has no pain and no quadriceps lag.

Case 4

This 60-year-old man had had his left knee fused for osteoarthritis 20 years before (Fig. 2a and b). He had undergone patellectomy at the same time. Subsequently severe osteoarthritis developed in the other knee, so the knee fusion was disassembled and, with an absent patella, a Guepar II hinge was used (Fig. 2c and d). At nine months his range of motion was from 0° to 70° without pain or quadriceps lag. His other knee will shortly be replaced by a prosthesis.

Discussion

The knee requires the presence of collateral ligaments for mediolateral stability and the cruciates or patellar mechanism for anteroposterior stability.[2,3] Hence, semiconstrained prostheses, I.C.L.H. and Tricon P were used in two patients whose patella was intact, and a hinge prosthesis in two patients with no patella.

Initially, I feared that the collateral

Fig. 2a

Fig. 2c

Fig. 2b

Fig. 2d

FIG. 2—Case 2. (a and b) Knee that was fused with patellectomy 20 years ago. (c and d) Guepar hinge prosthesis inserted. Very little bone resection was necessary and leg length was largely restored.

ligaments would be absent, but not only were they present as definable structures in all these patients, but they and the joint capsule could easily be separated from the underlying bone by means of a periosteal elevator. Fortunately, there is no technique of knee fusion that damages the collateral ligaments.

I also feared that because in most of these cases the quadriceps had not moved or functioned for many years, either no movement would be achieved or a quadriceps lag would result. Amazingly, this has not been so and no patient had a quadriceps lag at any time. The initial range of movement was frequently disappointing, being only from 0° to 30° for the first few weeks, but in all cases flexion slowly improved to at least 90° after 1 year.

The lesson learned from these four patients is that knee fusion disassembly is possible and does work. Patients should be reassured about the range of motion they will attain, and manipulation should not be carried out to regain flexion; it will come spontaneously with the passage of time.

The patella should not be resected at the time of knee fusion because it is necessary for anteroposterior stability if total knee replacement with a semiconstrained prosthesis must be carried out subsequently. Whether or not the patella is fused to the anterior femoral condyles is immaterial.

References

1. CAMERON HU, HARRIS WR: Acquired valgus instability after knee replacement. *Clin Orthop* 1981; 154: 216–219
2. CAMERON HU, FEDORKOW DM: The patella in total knee arthroplasty. *Clin Orthop* 1982; 165: 197–199
3. CAMERON HU: Antero-posterior instability in knee replacements. *Acta Orthop Belg* 1984; 50: 476–480

BOOK REVIEWS

ATLAS OF SKELETAL DYSPLASIAS. Ruth Wynne-Davies, Christine M. Hall and A. Graham Apley. 646 pp. Illust. Churchill Livingstone, Edinburgh; Academic Press Canada, Don Mills, Ont., 1985. $230.95. ISBN 0-443-03047-2.

This superbly illustrated atlas takes a fresh look at a subject often considered confusing and dry. Thanks to a clear and systematic arrangement, reading and comparing various disorders is easy and interesting. Each disease is presented in an identical fashion. After a short introductory paragraph the inheritance and frequency are stated, leading to a description of the clinical and radiologic features, with the various sites always presented in the same order. A short note on bone maturation and biochemistry is then followed by a more extensive discussion of the differential diagnosis, prognosis and complications, and finally a short list of references. Not only are the x-ray films arranged in anatomical sequence, but they also show the evolution of the disease with aging. This facilitates the use of the atlas as a diagnostic tool.

In addition to skeletal dysplasias the atlas illustrates those malformation syndromes that are characterized by structural defects of one or more bones and involve more than one system.

Although the atlas is primarily designed for orthopedic surgeons, radiologists, pediatricians, geneticists and allied health professionals, residents will profit greatly from this *chef-d'oeuvre.*

The use of this atlas could be enhanced by extending the list of references.

This publication fills a long-time void in the specialty literature and will be appreciated by the above-mentioned medical community and certainly should receive high praise internationally. In fact, if it is not already an indispensable standard book it will become so in the very near future.

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DISEASES OF THE AORTA Including an Atlas of Angiographic Pathology and Surgical Technique. E. Stanley Crawford and John L. Crawford. 401 pp. Illust. Williams & Wilkins, Baltimore, 1984. $135. (US). ISBN 0-683-02235-0.

At the outset, it should be stated that this is not a book for the occasional vascular surgeon. Information concerning pathophysiology, although lucid, is sketchy, and diagnostic aids and guidelines are not forthcoming. Preoperative preparation is not described in any detail, and intraoperative and postoperative management are left to the operating surgeon's experience and whim. With this established, one is left with a sense of wondrous admiration of the authors' attainment of their stated goal: "to present, in picture form, an atlas of all diseases of the aorta, their variations and the various surgical techniques available for their correction". It is unlikely that any surgeon (including fellow Baylor surgeon Michael DeBakey) can lay claim to the concentrated clinical experience of the senior author. He and his coauthor have provided a valuable service to all vascular surgeons by building upon their experiences to develop this volume. In 12 chapters, the basic clinical features, radiologic findings and surgical approaches to aortic disease are succinctly presented.

As befits its title as an atlas, this work should prove invaluable to those contemplating a career in cardiovascular surgery. Its cost may exclude it from every surgical resident's library, but it portrays the fascinating spectrum of conditions affecting the aorta. Developmental, degenerative, infectious and traumatic lesions are dealt with in turn, as are the more esoteric neoplastic conditions. The intricacies of repeat operations are also presented in some detail. In my opinion this atlas will help make the junior surgeon aware of aortic disease as a possible diagnosis and provide enlightenment regarding theoretical correction. At the other end of the scale, this publication offers to the established vascular surgeon a rich compendium of possible surgical approaches to aortic problems. It assumes that the surgeon has an awareness of cardiopulmonary bypass as well as profound hypothermia and then provides technical guidelines to the repair of a staggering number of aortic lesions likely to be encountered.

The atlas is extremely well organized. Each chapter is concisely arranged, making reference to a specific lesion, or its characteristics, simple. The illustrations by Perrin Smith evoke memories of Frank Netter (if such comparisons are not odious to both concerned). They are awesome in their clarity, if not their veracity, considering the conditions under which the surgeon often labours. The juxtaposition of illustration to roentgenogram is uniform and requires little effort to correlate throughout. Although it may appear churlish, several typographical errors are apparent, but detract little from the general low-key, extremely professional discussion of aortic disease and its correction.

In conclusion, this atlas is a fine effort by superb surgeons and is highly recommended.

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NORRIS AND CAMPBELL'S ANAESTHETICS, RESUSCITATION AND INTENSIVE CARE. 6th ed. Donald Campbell and Alastair A. Spence. 254 pp. Illust. Churchill Livingstone, Edinburgh; Academic Press Canada, Don Mills, Ont., 1984. Price not stated, paperbound. ISBN 0-443-0300-57.

The authors of this small introductory textbook for medical students and hospital residents have made extensive revisions to this sixth edition since it was last published in 1978. They state in the preface that their objectives are to explain and clarify the underlying principles of good, safe, anesthetic care within the operating room and the surgical intensive care unit. They also correctly emphasize that the specialty of anesthesia has much that is relevant and important to all doctors in training, and it is in this context that the book is being reviewed for a surgical journal.

To distil the essence of the whole specialty of anesthesia into a 250-page paperback book is not an easy task, but it is one that the authors have done remarkably well. It is clearly written, coherent and keeps its objectives in correct perspective. The chapters are short, pertinent and cover all the essential topics. These

continued on page 56

NICOLE LE SAUX, MD, FRCPC; GODFREY K.M. HARDING, MD, FRCPC, FACP

Bacillus cereus Endophthalmitis

The authors present a case of a young man with post-traumatic endophthalmitis caused by *Bacillus cereus*. The clinical course was typical of the panophthalmitis caused by this toxin-producing organism: rapid onset of signs of systemic infection, a corneal ring abscess and eventual loss of the globe requiring enucleation. Studies of experimental rabbit models of the infection have indicated that the most efficacious regimen consists of systemic and topical clindamycin and gentamicin. Successful therapy depends upon immediate systemic administration of these agents when the clinical setting suggests *Bacillus cereus* infection.

Les auteurs présentent le cas d'un jeune homme qui a développé une endophtalmite à *Bacillus cereus* après un traumatisme oculaire. L'évolution clinique était typique de la panophtalmite causée par cet organisme produisant une toxine: apparition rapide d'une atteinte systémique, présence d'un abcès cornéen, et nécessisté de procéder à une énucléation. L'antibiothérapie la plus efficace est la combinaison de clindamycine et de gentamicine, d'après les études d'un modèle expérimental chez le lapin. L'usage immédiat de ces antibiotiques par voie parentérale est essentiel lorsque le contexte clinique suggère une infection à *Bacillus cereus*.

Post-traumatic endophthalmitis occurs in approximately 2.4% of penetrating ocular injuries. *Bacillus cereus* endophthalmitis, although uncommon, is particularly devastating because of its virulence. We present a case of *B. cereus* endophthalmitis to illustrate its fulminant course. Physicians should consider this organism in the setting of traumatic eye injury in order that optimal antimicrobial therapy be instituted as quickly as possible.

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Case Report

A healthy 29-year-old man, while cutting spruce with a circular saw, experienced acute pain in the right eye after an object struck his nose. Within an hour he was examined, started on gentamicin and penicillin intravenously and transferred to our hospital. There, ocular examination of the affected eye revealed light perception vision and a nasal corneal limbal laceration with iris prolapse.

He underwent a primary surgical repair of the laceration. No intraocular foreign body was identified. Penicillin and gentamicin were continued postoperatively. Within 24 hours, he complained of increased ocular pain and had a temperature spike of 39°C. Examination 48 hours postoperatively revealed marked lid edema, chemosis, corneal ring abscess and purulent discharge at the operative site (Fig. 1). A diagnosis of endophthalmitis was made. An anterior chamber aspirate revealed pus cells and gram-positive bacilli (Fig. 2) which subsequently grew *B. cereus* in pure culture.

Although clindamycin and chloramphenicol were given in addition to gentamicin, the patient had no further improvement and required an enucleation 23 days after the injury.

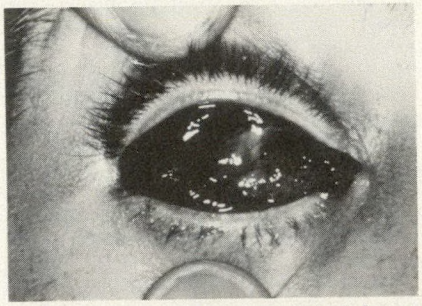

FIG. 1—Note marked conjunctival chemosis and corneal ring abscess.

Discussion

Bacillus cereus endophthalmitis is a relatively uncommon yet devastating complication of penetrating ocular trauma.[1-4] At the time of this report, there have been no reported cases in which useful vision was preserved after this infection. Although the pathogenicity of the *Bacillus* sp. was first noted by François in 1934,[5] the first positive bacteriologic identification of *B. cereus* causing endophthalmitis was published by Davenport and Smith in 1952.[1] Subsequently, these intraocular infections have occurred in two broad clinical settings. Most cases have been associated with bacteremia secondary to intravenous heroin

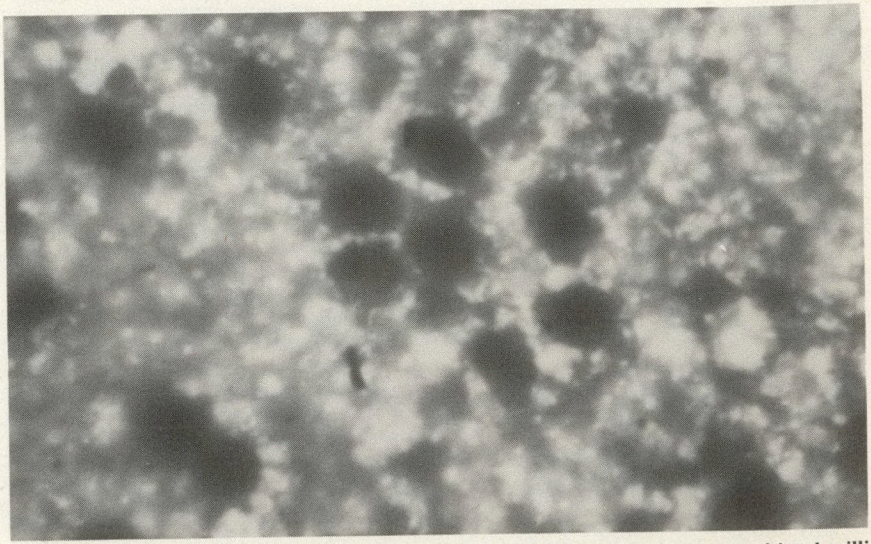

FIG. 2—Gram's staining of anterior chamber aspirate showing gram-positive bacilli consistent with *Bacillus cereus* (oil immersion, original magnification × 1250).

abuse. Cases associated with trauma, such as the one we report, are being recognized with increasing frequency. These usually occur in contaminated settings or in association with metal working tools.

The organism is an aerobic endospore-forming gram-positive bacillus that is ubiquitous in nature, being present in air, soil, water, milk, dust, wool, stool and normal conjunctival sacs. It has also been found in cultures of instruments used for intravenous injection of heroin.[2,6] Although usually sensitive to chloramphenicol and tetracycline, the lowest minimum inhibitory concentrations are to clindamycin and gentamicin. It is usually resistant to penicillins and cephalosporins.

The clinical course is unique when compared with other bacterial endophthalmitides. Pain, chemosis, fever and peripheral corneal edema predominate in the first 24 to 36 hours after injury. During this phase, there is rapid vitreous destruction caused by toxin release. Consequently, vision rapidly deteriorates. During the next 48 hours, a corneal ring abscess occurs. At this stage, there is leukocytosis, often with a left shift. Within 72 hours, there is irreversible blindness, secondary to a rapidly destructive panophthalmitis. Other infectious causes of endophthalmitis do not display such systemic manifestations nor such a fulminant clinical course.

The ultimate prognosis of *B. cereus* endophthalmitis is poor. The organism multiplies rapidly to release toxin, causing vitreal necrosis while the diagnosis is neither obvious nor considered. In 1981, O'Day and colleagues[3] studied an experimental rabbit model in order to clarify the pathogenesis and gain some insight into the management of the problem. Employing a combination of gentamicin and clindamycin systemically and periocularly, they found that, when used early in the course, it provided the best eradication of the organism from the globe. Tabbara and O'Connor[7] demonstrated excellent absorption of clindamycin phosphate by ocular tissue after parenteral or subconjunctival injection. It is unknown at present whether early empiric antibiotic therapy directed against *B. cereus* in a clinical setting would be successful in preserving vision. However, knowing that therapy of the established infection is futile, it would seem judicious to institute therapy with clindamycin and gentamicin in the setting of penetrating ocular trauma involving potential sources of *B. cereus*. Should the infection not become manifest within 48 to 72 hours of injury, then the antibiotics could be withdrawn. Vitreous and anterior chamber taps for Gram's staining and culture should be obtained by the ophthalmologist as soon as possible after injury.

In summary, we present a typical clinical case of *B. cereus* endophthalmitis in the setting of penetrating ocular trauma. Because of the dismal prognosis with established endophthalmitis, we recommend early empiric institution of intravenous and periocular clindamycin and gentamicin.

We thank Dr. Davinder Singh for involving us in the care of his patient.

References

1. DAVENPORT R, SMITH C: Panophthalmitis due to an organism of the *Bacillus subtilis* group. *Brit J Ophthalmol* 1952; 36: 389–392
2. HO PC, O'DAY DM, HEAD WS: Fulminating panophthalmitis due to exogenous infection with *Bacillus cereus:* report of 4 cases. *Br J Ophthalmol* 1982; 66: 205–208
3. O'DAY DM, SMITH RS, GREGG CR, et al: The problem of bacillus species infection with special emphasis on the virulence of *Bacillus cereus. Ophthalmology* 1981; 88: 833–838
4. YOUNG EJ, WALLACE RJ JR, ERICSSON CD, et al: Panophthalmitis due to *Bacillus cereus. Arch Intern Med* 1980; 140: 559–560
5. FRANÇOIS JM: Le bacille subtilique en pathologie oculaire. *Bull Mem Soc Fr Ophthalmol* 1934; 47: 423–437
6. SHAMSUDDIN D, TUAZON CU, LEVY C, et al: *Bacillus cereus* panophthalmitis: source of the organism. *Rev Infect Dis* 1982; 4: 97–103
7. TABBARA KF, O'CONNOR GR: Ocular tissue absorption of clindamycin phosphate. *Arch Ophthalmol* 1975; 93: 1180–1185

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The Hartmann Procedure

In 1923, Hartmann described an operation for cancer of the rectosigmoid area in high-risk patients not amenable to abdominoperineal resection.

In the period 1972 to 1984 at the Queen Elizabeth Hospital in Montreal interest in the Hartmann procedure, with resection, end-colostomy and suture closure of the distal colon intraperitoneally, was renewed. The main indication for the procedure was diverticulitis, with cancer second.

The author reviews the experience with 64 patients (average age 80 years) who underwent the Hartmann procedure. Obstruction, perforation and abscesses were the commoner indications. The mortality was 17%, but this included several patients who died from 1 to 6 months postoperatively of multiple-organ failure not necessarily related to the operation. A 30-day death rate was 8%. The complication rate was 35%. Restoration of bowel continuity (second stage) was done 2 to 3 months later in 28 cases with no deaths.

En 1923, Hartmann décrivit une opération des cancers de la région rectosigmoïdienne applicable aux patients à risque élevé qui ne peuvent subir une résection abdominopérinéale. Entre 1972 et 1984, à l'hôpital Reine Elizabeth de Montréal, on a repris intérêt dans l'opération de Hartmann avec résection, colostomie terminale et suture intrapéritonéale du côlon distal. La principale indication de cette opération est la diverticulite, le cancer venant en second.

L'auteur passe en revue une expérience portant sur 64 patients (dont l'âge moyen était de 80 ans) qui ont subi une intervention de Hartmann. L'obstruction, la perforation et les abcès ont été les principales indications. La mortalité a été de 17% mais ceci inclut plusieurs patients qui sont morts de 1 à 6 mois après l'opération, d'insuffisance plurifonctionnelle sans qu'il n'y ait nécessairement de lien avec l'opération. La mortalité à 30 jours a été de 8%. Le taux de complication a été de 35%. Le rétablissement de la continuité intestinale (dans un deuxième temps) a été affectué 2 à 3 mois plus tard dans 28 cas, sans qu'il n'y ait de décès.

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Henri Hartmann is best remembered for the operation he described in 1923,[1] a less-extensive procedure for cancer of the rectosigmoid colon than the usual abdominoperineal resection. It comprised excision of the sigmoid lesion with an end-colostomy and suture closure of the distal rectosigmoid area. By 1931 Hartmann had reported 34 cases with an 8% death rate.[2]

Because Hartmann's procedure has also been used to manage complications of diverticulitis,[3,4] I decided to review our experience with this operation at the Queen Elizabeth Hospital in Montreal.

Table I—Popularity of the Hartmann Procedure From 1972 to 1984

Year	No. of patients
1972	4
1973	3
1974	3
1975	1
1976	3
1977	3
1978	5
1979	5
1980	9
1981	5
1982	7
1983	8
1984	8

Clinical Material

The records of 64 patients who underwent Hartmann's procedure between 1972 and 1984 were examined retrospectively. Eight general surgeons, with resident staff, of a 285-bed community hospital were involved in their care.

Findings

Two groups of patients were seen — 34 patients who had resection only and 30 who had intestinal continuity restored

Table III—Indications for the Hartmann Procedure

Indication	No. of patients	
Diverticulitis		36
Perforation	23	
Obstruction	6	
Abscess	7	
Cancer		24
Recurrence	3	
Perforation	9	
Obstruction	7	
Palliation	3	
Diverticulitis	2	
Ischemia		2
Volvulus with gangrene	1	
Ischemic colitis	1	
Colitis		2
Crohn's disease	1	
Ulcerative colitis	1	

Table II—Age, Sex and Use of Hartmann Procedure, 1972 to 1984*

Patient age, yr	Resection						Restoration					
	Inflammation (diverculitis)			Cancer			Inflammation (diverticulitis)			Cancer		
	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
50–60	6	2	8	5	0	5	5	0	5	1	0	1
61–70	8	3	11	4	1	5	6	4	10	0	0	0
71–80	4	6	10	2	1	3	3	2	5	5	0	5
81–90	0	7	7	5	4	9	0	2	2	0	0	0
> 90	0	0	0	0	2	2	0	0	0	0	0	0
Totals	18	18	36	16	8	24	14	8	22	6	0	6

*2 patients with ischemia and 2 with colitis excluded.

after resection. The mean age of the 64 patients was 80 years.

Table I reveals the increasing popularity of the Hartmann procedure in our institution during the 12-year period and the greater frequency of its use to treat diverticulitis over cancer (Table II).

Among the indications for the Hartmann procedure (Table III), we noted that perforation was common in diverticulitis (64%), whereas obstruction (15% to 25%) was equal in cancer and diverticulitis. Abscess or phlegmon accounted for 28% of the diverticulitis group. Perforation occurred in 30% of the cancer group. Two patients had ischemic necrosis and two inflammatory bowel disease.

The majority of second-stage operations restoring bowel continuity involved those under 80 years of age (Table II). The average hospital stay after resection was 1 month; that after restoration of bowel continuity was such shorter (Table IV).

Complications

The complication rate for the Hartmann procedure was 35%; the majority of these complications occurred in patients with diverticulitis (Table V). Following restoration of bowel continuity the complication rate was 46% (Table VI), but none of the complications were fatal. Many recent reports of the Hartmann procedure refer to the technical advantage of staplers in restoring continuity, but in our series hand suturing was used, in almost all cases, with satisfactory healing and only one possible leak.

Deaths

Eleven (17%) of the 64 patients died after resection. Of the 49 survivors, 28 subsequently had bowel continuity restored an average of 4 months later in cases of diverticulitis and 2 months later for cancer patients. No patients who underwent restoration of bowel continuity died. The deaths were divided equally between those with cancer and diverticulitis (Table VII). Six deaths occurred from 1 to 6 months postoperatively, due to a variety of other diseases. Were we to calculate the death rate within 30 days of operation, the figure of 8% would be comparable to that of Nunes and associates[5] reporting on 25 cases of diverticulitis.

Table IV—Duration of Hospitalization After Hartmann Procedure and Restoration of Bowel Continuity*

	Diverticulitis	Cancer	Ischemia
Hartmann procedure, no.	36	24	2
Hospital stay, d			
Range	10 – 120	12 – 90	19 – 40
Average	30	25	30
Deaths	4	6	1
Survivors	32	18	1
Colostomy closure	22	6	
Time between stages, d			
Range	50 – 120	30 – 120	
Average	120	70	
Hospital stay, d			
Range	11 – 33	12 – 60	
Average	19	18	

*2 patients with colitis excluded.

Table V—Complications of Hartmann Procedure in 49 Survivors

Complication	No. of patients	
	Cancer	Diverticulitis
Rectal stump abcess	1	
Colostomy stenosis		3
Colostomy necrosis		2
Wound infection		4
Small-bowel resection		1
Pulmonary embolism		1
Incisional hernia	1	1
Psychosis		1
Dehiscence		2

Table VI—Complications in 28 Patients Who Underwent Restoration of Bowel Continuity

Complication	No. of patients
Small-bowel injury	2
Small-bowel resection required	1
Splenic injury	1
Leak of anastomosis	1
Wound infection	4
Stroke	1
Incisional hernia	2
Subphrenic abscess	1

Table VII—Deaths After Hartmann Procedure

Diagnosis	Age, yr	Cause of death
Diverticulitis	88	Senility, 42 d "allowed to die"
Diverticulitis	76	Brain cancer metastases, lung primary, 211 d
Diverticulitis	93	Sepsis, 2 d
Cancer	78	Depression; melena; pneumonia; gastrectomy, 60 d
Cancer	90	Multiple-organ failure, 90 d
Cancer	62	Respiratory failure, lung cancer, 3 d
Volvulus	95	Multiple-organ failure, 6 mo
Cancer	56	Gas gangrene, thigh, 3 mo
Diverticulitis + cancer	71	Recurrent obstruction, 42 d
Cancer + diverticular abscess	73	Necrotizing fasciitis, 5 d
Cancer	60	Perforation, 15 d

Table VIII—Indications for the Hartmann Procedure Other Than Cancer

Diverticulitis sigmoid colon
Cancer sigmoid colon
Volvulus sigmoid colon
Traumatic perforation (colonoscopy)
Ischemic lesions
Radiation injuries
Anastomotic disruption after anterior resection
Inadequately prepared bowel

Comment

The use of a tube to identify the rectal stump and of an end-to-side coloproctostomy have facilitated the operation. The main hazards in restoring continuity have been injury to the small bowel, due to adhesions, or to the spleen in mobilizing the splenic flexure.

The Hartmann procedure was revived for indications other than cancer (Table VIII). Some surgeons continue to risk a one-stage primary anastomosis which, if unsuccessful, may then require a Hartmann procedure.

Surgery for acute complications of lower left colonic disease has undergone many changes.

A review of our 64 cases revealed that this procedure is good for the severe complications of left colonic disease. The high complication rate is not surprising in view of the severity of the illness and the age of patients at its onset. Most patients came to the hospital with an acute abdomen and received operative treatment on the day of admission. The resulting death rate was 17%.

References

1. HARTMANN H: Note sur un procédé nouveau d'extirpation des cancers de la partie terminale du côlon. *Bull Mem Soc Chir Paris* 1923; 49: 1474–1477
2. MARQUAND J, GUIVARC'H M, GARAT P, et al: [The place of Hartmann's operation in the treatment of complicated lesions of the descending colon. 58 Hartmann resections including 43 as an emergency.] *J Chir (Paris)* 1979; 116: 161–166
3. BAKKER FC, HOITSMA HF, DEN OTTER G: The Hartmann procedure. *Br J Surg* 1982; 69: 580–582
4. LUBBERS EJ, DE BOER HH: Inherent complications of Hartmann's operation. *Surg Gynecol Obstet* 1982; 155: 717–721
5. NUNES GC, ROBNETT AH, KREMER RM, et al: The Hartmann procedure for complications of diverticulitis. *Arch Surg* 1979; 114: 425–429

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La palliation du cancer de l'oesophage par prothèse endoluminale

De 1973 à 1984, 27 patients présentant un cancer de l'oesophage ou du cardia et souffrant de dysphagie ont bénéficié d'une palliation par prothèse endoluminale. L'intubation, effectuée sous oesophagoscopie ou à travers une gastrotomie, ou les deux, a permis de placer 23 tubes de Célestin et 4 prothèses de Mousseau–Barbin. La mortalité opératoire a été de 11%. La morbidité globale a été de 37%, caractérisée par des complications telles que migration du tube (18.5% des patients), dyspnée par mauvais placement du tube (7.4%), obstruction de la prothèse (11%), fistule oesophagienne (7.4%), aspiration (7.4%) et suppuration de la plaie opératoire (7.4%). Le passage alimentaire a été d'emblée satisfaisant chez 24 patients.

Between 1973 and 1984, 27 patients with a cancer of the esophagus or the cardia and suffering dysphagia underwent palliation with an endoluminal prosthesis. Intubation under esophagoscopy or through a gastrotomy, or both, allowed the placement of 23 Celestin tubes and 4 Mousseau–Barbin prostheses. The early postoperative death rate was 11%. The overall morbidity was 37%. It included such complications as tube displacement (18.5% of patients), tracheal compression by the tube (7.4%), obstruction of the prosthesis (11%), esophageal fistula (7.4%), aspiration (7.4%) and wound infection (7.4%). The ability to swallow was acceptable in 24 patients.

La dysphagie est le maître-symptôme dans le cancer de l'oesophage et du cardia.[1,2] Sa palliation fait appel à la chirurgie, l'intubation endoluminale, la radiothérapie, la chimiothérapie et le laser; ce dernier, plus récent, semble très prometteur.[3] La chirurgie palliative consiste à réaliser des courts-circuits. Elle demeure la meilleure méthode quant à la qualité de la palliation.[4-6] Mais, pratiquée sur des sujets dénutris, elle est grevée d'un taux de mortalité prohibitif.[7-10] La chimiothérapie et la radiothérapie sont des méthodes souvent adjuvantes à la chirurgie ou à l'intubation.[11,12]

La première intubation oesophagienne aurait été pratiquée par d'Etiolles en 1845.[13] En 1924, Souttar crée un tube métallique flexible dont l'installation se fait à travers un oesophagoscope rigide par la bouche, c'est la méthode dite de pulsion (''push-through'').[13] En 1956, Mousseau et collaborateurs[14] inventent un tube en néoplex qui est introduit par la bouche et tiré à travers une gastrotomie, c'est la technique de traction (''pull-through''). En 1959[15] puis en 1969,[16] Célestin améliore le modèle de Mousseau-Barbin. En 1977, Atkinson et Ferguson[17] inventent une technique de pose, à l'aide d'un fibroscope, d'un tube de Célestin modifié.[18] Il existe actuellement une trentaine de tubes endo-oesophagiens, mais les plus utilisés sont ceux de Célestin et de Proctor-Livingstone.

De 1973 à 1984, 113 cancers de l'oesophage ont été traités à l'hôpital Maisonneuve-Rosemont; 36 patients (32%) ont bénéficié d'une chirurgie d'exérèse, 3 sujets (3%) ont subi une chirurgie palliative. La radiothérapie isolée a été pratiquée chez 33 patients (29%). Treize malades (12%) sont décédés avant d'avoir reçu un quelconque traitement. Enfin, 27 patients (24%) qui font l'objet de notre étude ont bénéficié de l'intubation chirurgicale.

Patients et Méthode

Patients

De 1973 à 1984, palliation par intubation oesophagienne a été effectuée chez 27 patients porteurs de cancer de l'oesophage ou du cardia et souffrant de dysphagie; 23 fois on a utilisé un tube de Célestin et 4 fois une prothèse de Mousseau–Barbin. Nous comptons 22 hommes et 5 femmes dont l'âge moyen est de 65 ans (variant de 40 à 85 ans). Le plus grand nombre se retrouve au-delà de 60 ans (19 cas). La symptomatologie a été dominée par la dysphagie retrouvée constamment et qui a été l'indication de l'intubation oesophagienne. Une intubation a été pratiquée dans un cas de fistule trachéo-oesophagienne. Nous avons trouvé, associées au cancer, certaines maladies comme le diabète (quatre fois), l'hypertension artérielle (trois fois), le cancer du pancréas (une fois).

La localisation la plus fréquente du cancer est au tiers inférieur de l'oesophage (14 cas), suivi du tiers moyen (6 cas), du cardia (4 cas), de l'union du tiers moyen et tiers inférieur (2 cas) et enfin du tiers supérieur (1 cas).

Les types histologiques rencontrés sont le cancer épidermoïde (14 cas), l'adénocarcinome (12 cas) et d'épithélioma mal différencié (1 cas). Nous dénombrons neuf cas de métastases évidentes au moment de l'intervention.

Sélection des patients

Le diagnostic du cancer de l'oesophage est posé sur le transit baryté, l'endoscopie et la biopsie. Divers critères ont prévalu quant au choix de la technique d'intubation chirurgicale. Ces critères ont évolué au cours des années, parallèlement à l'affinement du matériel et des méthodes, notamment celles de l'anesthésie. Les différents éléments de l'indication d'une intubation chirurgicale sont les suivants:

- Présence d'une dysphagie importante.

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- Impossibilité de réséquer la tumeur de l'oesophage du fait de l'extension locale ou des métastases.
- Âge avancé du patient, présence d'un mauvais état général ou d'une tare majeure, tous facteurs proscrivant une chirurgie majeure.
- Refus par le patient ou sa famille d'une intervention majeure à visée radicale.
- Présence d'une fistule aéro-oesophagienne.
- Inopérabilité de la tumeur établie au cours d'une laparotomie ou d'une thoracotomie.

Cette dernière éventualité représente actuellement la majeure partie de nos indications d'intubation chirurgicale. Sinon, les autres cas sont maintenant justiciables d'une intubation per endoscopique ou d'une application du laser.

Méthode

Matériel d'intubation.—Le tube de Mousseau-Barbin a été utilisé quatre fois. C'est un tube en néoplex. L'extrémité proximale est un entonnoir long de 40 mm et de 24 mm de diamètre. Le tube lui-même est long de 30 cm, d'un diamètre de 10 à 14 mm. La partie distale, longue de 70 cm et de 5 mm de diamètre, est effilée et tient lieu de bougie que l'on passe à travers la tumeur et par laquelle on tire le tube.

Le tube modifié de Célestin, encore appelé tube série IV, a été utilisé 23 fois. Il est fait en latex sur une armature de nylon. La longueur du tube est de 28.5 cm avec un diamètre de 1.5 cm. La partie proximale, en forme de tulipe, est longue de 3.5 cm et possède un diamètre de 2.8 cm. Une bougie-pilote démontable, d'une longueur de 60 cm et d'un diamètre de 4.5 mm, est utilisée pour négocier la sténose.

Technique d'intubation.—L'intubation se pratique sous anesthésie générale. On effectue une laparotomie médiane susombilicale par laquelle on pratique une gastrotomie verticale de 3 cm sur la face antérieure de l'estomac, près du cardia. Dans certains cas faciles, la bougie-pilote reliée au tube, introduite par la bouche, passe la sténose et est saisie à travers la gastrotomie. Dans d'autres cas plus complexes, on négocie le passage de la sténose néoplasique par en bas, à travers la gastrotomie, avec un tube de Levin, une bougie de Jackson ou de Hurst ou une broche-guide dont l'extrémité est amarrée bout à bout avec du fil de soie à la bougie solidaire du tube introduite par la bouche. La traction du tuteur vers l'estomac, à travers la gastrotomie, permet de placer le tube. On vérifie avec l'endoscope que l'extrémité proximale du tube est épaulée par la tumeur. On laisse le tube dépasser de 3 cm dans l'estomac et on en coupe l'excès. Un point total à la soie 00 fixe le tube à la paroi gastrique. On procède à la fermeture de la gastrotomie et de la paroi abdominale. Une sonde de Levin, passant dans le tube, draine l'estomac jusqu'à la reprise de l'alimentation orale qui a lieu après 3 jours. On procède à une vérification radiologique de la position du tube après l'intervention et avant la reprise de l'alimentation orale.

Traitement associé.—La radiothérapie, souvent utilisée comme premier choix thérapeutique, a été effectuée chez 13 patients. La chimiothérapie a été réalisée deux fois.

Resultats

Mortalité

Il n'y a pas eu de décès peropératoire. La mortalité opératoire à 30 jours est de 11% (3 sur 27). L'un des patients est décédé au 4e jour dans un tableau d'insuffisance respiratoire aiguë. Cette complication a été provoquée par la compression de l'épiglotte par le bout proximal du tube et par une broncho-pneumopathie par aspiration massive.

Un autre patient a présenté également une aspiration massive. Le décès est survenu au 15e jour postopératoire dans un tableau d'infection pulmonaire et d'insuffisance respiratoire aiguë.

Le 3e décès survenu au 15e jour postopératoire est imputable à un accident vasculaire cérébral.

Morbidité

Neuf patients (33%) ont présenté des complications notables, relativement imputables à la technique.

Nous avons constaté deux cas de dyspnée en rapport avec un mauvais placement du tube dont le bout proximal se trouvait au niveau de l'épiglotte. Deux cas d'aspiration massive (7.4%) déjà mentionnés, dont l'un associé à une dyspnée par compression de l'épiglotte, ont eu une issue fatale. Une obstruction alimentaire a été traitée en changeant le tube.

La complication à distance la plus fréquent demeure la migration. Nous en comptons cinq cas (18% des patients). Deux cas ont été accompagnés d'une obstruction alimentaire, proximale dans un cas et distale dans l'autre. Deux fistules oesophagiennes (7.4% des patients), l'une trachéobronchique et l'autre borgne, étaient associées à une migration du tube. Toutes ces complications en rapport avec une migration se sont amendées avec l'ablation et le repositionnement du tube. Des régurgitations significatives ont été présentés dans quatre cas (15%).

Le passage alimentaire a été jugé convenable chez tous les patients, notamment dans les cas d'obstruction traités par changement (un cas) ou positionnement du tube (deux cas). Cette palliation permet l'alimentation aux liquides et aux aliments passés au mixeur.

Le séjour hospitalier a été en moyenne de 28 jours (écart de 11 à 100 jours). Après mise en place du tube, la survie moyenne a été de 14 semaines (écart de 1 semaine à 2 ans).

Au total, avec cette méthode d'intubation chirurgicale, nous avons relevé 22 retours à la maison, 3 décès opératoires et 2 décès à l'hôpital non liés à l'opération.

Discussion

L'intubation chirugicale, un geste bénin, comporte toutefois quelques complications notamment liées au terrain particulier. Le risque anesthésique chez ce type de patient a souvent été incriminé; nous n'avons constaté aucun décès peropératoire.

Nous avons relevé une mortalité opératoire de 11% (3 sur 27). Cette valeur est faible, comparée aux chiffres fournis par Lishman et collaborateurs (45%),[19] Ammann et Collis (31%),[20] Diamantes et Mannell (25%),[21] avec l'intubation chirugicale. Notre taux de mortalité est grossièrement comparable à celui de la série de Wang et collaborateurs (8.1%)[2] et de Tata (12%).[22] Il est relativement du même ordre que celui qu'on trouve avec la méthode endoscopique (6% à 11%)[23] et parfois même plus bas.[21,24–27] On oppose également les méthodes chirurgicales et endoscopiques quant à la morbidité.

Dix patients (37%) ont présenté des complications directement en rapport avec la technique. Ce taux est supérieur aux valeurs fournies par Wang et collaborateurs (15%)[2] et Girardet et collaborateurs (26.5%),[13] mais il est du même ordre que les chiffres de Haynes et collaborateurs (79%),[28] de Angorn (39%),[29] de Diamantes et Mannell (34%)[21] avec la même méthode chirurgicale.

Nous avons relevé deux cas de dyspnée survenus pendant la période postopératoire immédiate, du fait d'une compression de l'épiglotte par le bout proximale d'un tube placé trop haut. L'un de ces patients qui présentait par ailleurs une aspiration massive est décédé 4 jours après l'opération dans un tableau d'insuffisance respiratoire aiguë. Cette complication, plus fréquente avec les tumeurs haut situées, disparaît habituellement avec le positionnement du tube.[1,13,28]

Cinq cas (18.5%) de migration du tube ont été enregistrés. Ces déplacements du tube étaient associés à deux obstructions alimentaires et deux fistules oesophagiennes dont l'une communiquait avec la trachée. Le taux de migration dans l'intubation chirurgicale est de 2.6% pour Saunders,[8] 5.3% pour Girardet et collaborateurs,[13] 29% pour l'équipe de

Haynes[28] et 2.5% pour celle de Wang et collaborateurs.[2] La migration est une complication moins fréquente dans l'intubation chirurgicale[5,19,29,30] et même dans les cas où le tube n'est pas fixé l'estomac.[31] Une bonne prévention de ces déplacements est assurée par la fixation de l'extrémité inférieure du tube à l'estomac par du fil non résorbable. Ces déplacements du tube ont été associés à deux obstructions alimentaires. L'occlusion de la partie supérieure du tube a été à l'origine de ces deux cas d'obstruction qui ont été traités par positionnement du tube. Un autre cas d'obstruction est survenu précocement, provoqué par l'impaction du bol alimentaire dans un tube de calibre trop petit. Ce dernier cas a été traité par changement du tube. L'obstruction est survenue avec une fréquence de 11%, qui entre dans le créneau des valeurs fournies par différents auteurs: de 2.7% à 29%.[2,8,20,28] Deux fistules (7.4%), borgne et tracho-oesphagienne, étaient associées à la migration du tube. Ces fistules, en rapport avec le génie propre de la maladie néoplasique, sont également dues à la nécrose par pression provoquée par le tube;[1] Tata[22] en relève 2 sur 34 patients. Nos deux cas ont nécessité la mise en place convenable d'un tube de calibre plus gros. Ce même traitement a été appliqué à un patient venu d'emblée avec une fistule tracho-oesophagienne. En effet, le traitement des fistules oesophagiennes en général et trachéo-bronchiques en particulier, relève de l'intubation ou de la fermeture chirurgicale.[32] Chez ces patients débilités, vus souvent au stade terminal, l'intubation est entaché d'une mortalité bien moindre et assure assez convenablement l'oblitération de la fistule.[2,29,32]

L'aspiration est survenue chez deux patients (7.4%) et l'on compte cependant quatre patients sujets à des régurgitations. Chaque fois que la prothèse franchit le cardia, le sphincter inférieur de l'oesophage est rendu incompétent et le reflux gastro-oesophagien devient inévitable avec son risque de pneumopathie par régurgitation.[4] Les broncho-pneumopathies d'aspiration sont survenues plus fréquemment dans d'autres séries (de 28.4% à 32.7%).[8,28,33] On peut expliquer la fréquence relativement faible de l'aspiration dans notre série par les mesures prophylactiques préconisées, notamment la surélévation de la tête du lit après la mise en place du tube.[2] C'est sans doute cela qui fait dire à certains auteurs que la crainte du reflux gastro-oesphagien, après intubation, est exagérée.[20]

Nous avons relevé deux suppurations pariétales (7.4%) taries au bout d'une quizaine de jours. Cette complication propre à l'intubation chirurgicale peut survenir jusque dans 50% des cas.[2,8,22,33,34] Nous n'avons pas relevé d'autres complications notables.

La survie moyenne de nos patients est de 14 semaines (entre 1 semaine et 2 ans), comparable aux résultats colligés par Girardet et collaborateurs (4.2 mois),[13] Wang et collaborateurs (156.4 jours)[2] et Angorn (5.9 mois)[29] avec l'intubation chirurgicale. Nos résultats sont également comparables à ceux de Sarr et collaborateurs (3.2 mois)[27] qui utilisent la méthode endoscopique. Il faut remarquer que le choix de la technique n'influe pas tellement sur la durée de la survie, mais plutôt sur la qualité de celle-ci. L'avantage certain de la méthode endoscopique est chercher dans sa durée d'hospitalisation plus courte, 3 à 7 jours.[23,27,29] La durée moyenne d'hospitalisation pour les patients d'Angorn[29] ayant subi l'intubation chirurgicale est de 15.4 jours. En ce qui nous concerne, nous avons une durée d'hospitalisation moyenne de 28 jours, plus grande que les valeurs précitées. L'explication en est que l'on garde plusieurs de nos patients à l'hôpital afin notamment d'améliorer leur état général souvent très altéré.

Conclusions

L'étude de ce groupe de 27 patients porteurs de cancer de l'oesophage ou du cardia nous permet de dire que la mise en place chirurgicale de prothèse endoluminale est relativement simple, possède une mortalité et une morbidité acceptables, assez comparable en cela à la méthode d'intubation par pulsion, assure une bonne palliation de la dysphagie, améliorant ainsi le confort de la survie. Nous considérons que l'intubation chirurgicale demeure encore une bonne indication dans les dysphagies néoplasiques et doit conserver sa place dans l'arsenal des méthodes palliatives du cancer de l'oesophage.

Références

1. EARLAM R, CUNHA-MELO JR: Malignant oesophageal strictures: a review of techniques for palliative intubation. *Br J Surg* 1982; 69: 61–68
2. WANG PY, YEH TJ, CHEN CL, et al: A spiral-grooved endoesophageal tube for management of malignant esophageal obstruction. *Ann Thorac Surg* 1985; 39: 503–507
3. FLEISCHER D, KESSLER F, HAYE O: Endoscopic Nd:YAG laser therapy for carcinoma of the esophagus: a new palliative approach. *Am J Surg* 1982; 143: 280–283
4. HAY JM, BOUDINET A: [Palliative treatment of cancer of the esophagus]. *Rev Prat* 1984; 34: 1777–1778, 1781–1782, 1785–1786
5. WATSON A: A study of the quality and duration of survival following resection, endoscopic intubation and surgical intubation in oesophageal carcinoma. *Br J Surg* 1982; 69: 585–588
6. MANNELL A: The palliation of esophageal cancer. *Surg Annu* 1985; 17: 249–270
7. EARLAM R, CUNHA-MELO JR: Oesophageal squamous cell carcinoma: I. A critical review of surgery. *Br J Surg* 1980; 67: 381–390
8. SAUNDERS NR: The Celestin tube in the palliation of carcinoma of the oesophagus and cardia. *Br J Surg* 1979; 66: 419–421
9. EL-DOMEIRI A, MARTINI N, BEATTIE EJ JR: Esophageal reconstruction by colon interposition. *Arch Surg (Chicago)* 1970; 100: 358–362
10. POSTLETHWAIT RW, SEALY WC, DILLON ML, et al: Colon interposition for esophageal substitution. *Ann Thorac Surg* 1971; 12: 89–109
11. MANNELL A: Carcinoma of esophagus. *Curr Probl Surg* 1982; 19: 553–647
12. WERNER ID: The multi-disciplinary approach in the management of squamous carcinoma of the oesophagus: the Groote Schuur Hospital experience: September 1971–September 1976. *Front Gastrointest Res* 1979; 5: 130–135
13. GIRARDET RE, RANSDELL HT JR, WHEAT MW JR: Palliative intubation in the management of esophageal carcinoma. *Ann Thorac Surg* 1974; 18: 417–430
14. MOUSSEAU M, LE FORESTIER J, BARBIN J, et al: Place de l'intubation à demeure dans le traitement palliatif du cancer de l'oesophage. *Arch Mal App Digest* 1956; 45: 208–214
15. CELESTIN LR: Permanent intubation in inoperable cancer of the oesophagus and cardia; a new tube. *Ann R Coll Surg Engl* 1959; 25: 165–170
16. Idem: Improvements in the Celestin tube for endo-oesophageal intubation in carcinoma and strictures. *Armamentarium* 1969; 5: 10
17. ATKINSON M, FERGUSON R: Fibreoptic endoscopic palliative intubation of inoperable oesophagogastric neoplasms. *Br Med J* 1977; 1: 266–267
18. ATKINSON M, FERGUSON R, PARKER GC: Tube introducer and modified Celestin tube for use in palliative intubation of oesophagogastric neoplasms at fibreoptic endoscopy. *Gut* 1978; 19: 669–671
19. LISHMAN AH, DELLIPIANI AW, DEVLIN HB: The insertion of oesophagogastric tubes in malignant oesophageal strictures: endoscopy or surgery? *Br J Surg* 1980; 67: 257–259
20. AMMANN JF, COLLIS JL: Palliative intubation of the esophagus. Analysis of 59 cases. *J Thorac Cardiovasc Surg* 1971; 61: 863–869
21. DIAMANTES T, MANNELL A: Oesophageal intubation for advanced oesophageal cancer: the Baragwanath experience 1977–1981. *Br J Surg* 1983; 70: 555–557
22. TATA HR: Experience with the use of the Mousseau-Barbin tube for palliation in carcinoma of the lower 2/3rd of the oesophagus. *Indian J Cancer* 1980; 17: 97–101
23. WATSON A: Palliative intubation in inoperable esophageal neoplasms (E). *Ann Thorac Surg* 1985; 39: 501–502
24. JONES DB, DAVIES PS, SMITH PM: Endoscopic insertion of palliative oesophageal tubes in oesophagogastric neoplasms. *Br J Surg* 1981; 68: 197–198
25. ROSE JD, SMITH PM: Fibre endoscopic insertion of palliative oesophageal tubes with the Nottingham introducer. *J R Soc Med* 1983; 76: 266–268
26. HEGARTY MM, ANGORN IB, BRYER JV, et al: Pulsion intubation for palliation of carcinoma of the oesophagus. *Br J Surg* 1977; 64: 160–165
27. SARR MG, HARPER PH, KETTLEWELL MG: Peroral pulsion intubation of malignant esophageal strictures using a fiberoptic technique. *Am Surg* 1984; 50: 437–440
28. HAYNES JW, MILLER PR, STEIGER Z, et al: Celestin tube use: radiographic manifestations of associated complications. *Radiology* 1984; 150: 41–44
29. ANGORN IB: Intubation in the treatment of carcinoma of the esophagus. *World J Surg* 1981; 5: 535–541
30. HEGARTY MM, ANGORN IB, BRYER JV, et al: Palliation of malignant esophago-respiratory fistulae by permanent indwelling prosthetic tube. *Ann Surg* 1977; 185: 88–91
31. HOLDEN MP, WOOLER HH, IONESCU MI: Mousseau-Barbin tubes for the treatment of carcinoma of the lower two-thirds of the oesophagus. Results and operative techniques. *Br J Surg* 1973; 60: 401–402
32. DURANCEAU A, JAMIESON GG: Malignant tracheoesophageal fistula. *Ann Thorac Surg* 1984; 37: 346–354
33. DAS SK, JOHN HT: Oesophageal intubation in obstructive lesions of the oesophagus. *Br J Surg* 1973; 60: 403–406
34. JOHNSON IR, BALFOUR TW, BOURKE JB: Intubation of malignant gastro-oesophageal strictures: a modification of the Mousseau-Barbin technique. *J R Coll Surg Edinb* 1976; 21: 225–228

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Allografts in Orthopedic Surgery: a Case Report and Literature Review

Recent advances in orthopedic surgery have reawakened interest in the use of osteochondral allografts. A case is presented of a 32-year-old man who was spared a hemipelvectomy for a huge chondrosarcoma of the pelvis by receiving a massive pelvic allograft. This is apparently the first report of such a procedure being performed in Canada. A history of allografting as well as the fate and immunologic aspects of bone grafts are presented. Bone-banking procedures and the clinical application of allografts in 1986 are discussed.

Les récents progrès dans le domaine de l'orthopédie ont renouvellé l'intérêt envers l'utilisation des allogreffes ostéochondrales. Le cas d'un homme de 32 ans chez qui une hémipelvectomie pour un énorme chondrosarcome du bassin a pû être évitée grâce à une allogreffe massive est ici rapporté. Il s'agit du premier cas du genre rapporté au Canada. Un historique des allogreffes et de leur évolution est présentée avec considération de l'aspect immunologique des greffes osseuses. L'application clinique des allogreffes et les procedures impliquant une banque d'os sont également discutées.

Allograft surgery is a rapidly growing area of knowledge and has numerous clinical applications that have not been widely reported in the surgical literature. For this reason and because multidisciplinary surgical teams are often required for major allograft surgery, we describe what we believe is the first report of such a procedure in Canada.

Case Report

A 30-year-old man complained to his family physician of an enlarging mass in the left lower quadrant of his abdomen. He had multiple exostoses, an autosomal dominant inherited disorder in which 20% of patients suffer malignant degeneration in one of the osteocartilaginous exostoses. A biopsy performed elsewhere confirmed the impression that the lesion was a huge chondrosarcoma of the pelvis (Fig. 1). Hemipelvectomy and ablation of the left lower extremity were recommended. The patient refused this mutilating procedure and came to our centre for consultation.

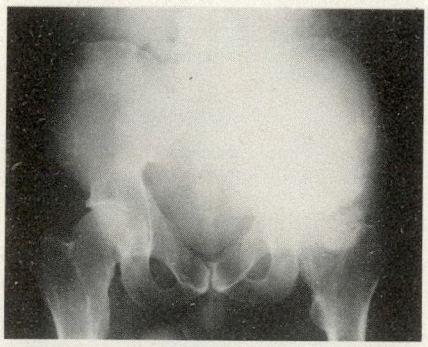

FIG. 1—Patient's pelvis showing huge chondrosarcoma involving left hemipelvis.

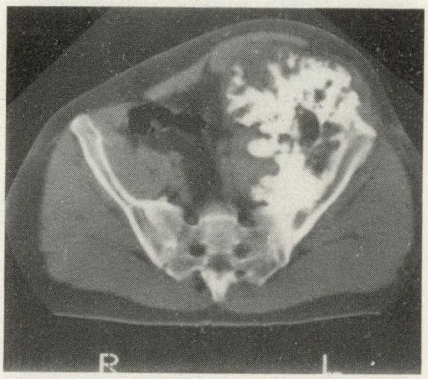

FIG. 2—Computerized tomogram of patient's pelvis demonstrating tumour.

After a thorough investigative work-up (Figs. 2 to 4) and review of the biopsy slides to confirm the diagnosis (Fig. 5), we proposed that a hemipelvic allograft be performed, preserving the lower extremity. The patient agreed in spite of the risks, and a search was undertaken for a suitable cadaver pelvis. A 45-year-old man in previous good health died suddenly of a massive myocardial infarction and was brought to our emergency department. The family gave permission to use the left hemipelvis which was immediately harvested using strict sterile technique in the operating room (Fig. 6). Deep freezing at -70°C was carried out to decrease the host allograft immune response.

We consulted with our colleagues in general surgery, urology and vascular surgery to plan the operation. It was begun by the urologic surgeon who catheterized the displaced left ureter to facilitate surgical identification during the procedure. The vascular surgeon mobilized the major vessels without violating the tumour. The orthopedic team then exposed and dislocated the hip joint, osteotomized the pelvis anteriorly and posteriorly and excised the tumour including the acetabulum and previous biopsy tract (Fig. 7). The allograft hemipelvis was thawed in saline, trimmed to match the defect, inserted and fixed with cancellous screws posteriorly and a large pelvic plate anteriorly. The femoral head was reduced

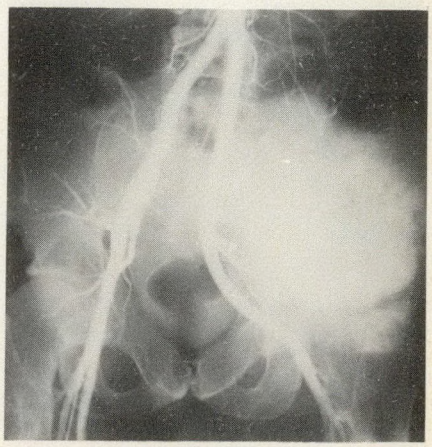

FIG. 3—Arteriogram demonstrating displacement of major vessels by tumour.

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and fitted perfectly. The anticipated prosthetic arthroplasty was not required. Soft tissues about the pelvis were reattached and reconstructed and the wound was closed in the usual fashion. A hip spica was applied and a postoperative roentgenogram revealed that the allograft was in good position (Fig. 8).

The patient underwent extensive rehabilitation to strengthen the musculature around the left hip joint and improved his gait pattern. Twelve months postoperatively he walked well without support and had returned to his preoperative activity level and life-style. Progressive degenerative changes in the left hip joint are anticipated and a painless ankylosis of the hip joint may be the ultimate result.

Discussion

The history of allograft surgery can be traced back to antiquity. In the third century AD, twin brothers, Cosmos, a physician, and Damian, a surgeon, performed many unusual medical feats. According to legend, these patron saints of allograft surgery resected the tumorous leg of a faithful church warden while he was asleep and replaced it with a portion of the lower limb from a Moor who had died the same day.[1]

Macewen in 1887 was the first to implant successfully a fresh bone allograft to replace a 10.8-cm defect in a humeral shaft caused by osteomyelitis.[2] The patient was a 3-year-old boy and the donor a patient who had undergone a corrective wedge osteotomy for "anterior tibial curves". In 1908, the German surgeon Erich Lexer published his series of 23 whole and 11 hemijoint allograft implants about the knee and, in a subsequent publication, claimed a 50% success rate.[1] Various other case reports followed, but interest in bone allografts was reawakened in the 1960s when discoveries by several researchers showed that the immunogenicity of bone graft can be reduced by freezing.[3]

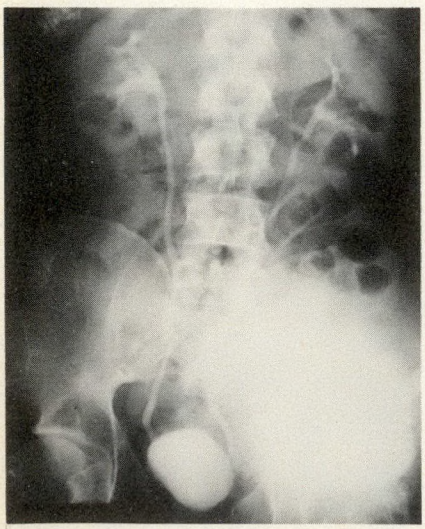

FIG. 4—Intravenous pyelogram shows displacement of left ureter by tumour.

Fate of Bone Grafts

The function of a bone graft is to stimulate osteogenesis and to provide structural support to that area of the body into which it is implanted. The osteogenic reparative process that the body uses to integrate the graft is known as incorporation. Researchers have shown that bone *autograft* incorporation begins with a host inflammatory response of partial necrosis and subsequent resorption of the implanted graft. This resorption is carried out by osteoclasts derived from circulating monocytes. There follows a vascular invasion, and deposition of new bone on dead bone begins by newly differentiated osteoblasts. This process is known as creeping substitution and continues until all the nonviable graft is replaced by newly formed bone. It is important to note that all of the implanted bone graft may not die. Portions of the graft remain viable from the nourishment it receives by peripheral diffusion or revascularization by microanastomosis.[4]

The incorporation of bone *allograft* is similar to the process described above but the vascular invasion is delayed and the allograft is never completely replaced by newly formed bone. These temporal and quantitative differences can be accounted for by the degree of genetic disparity between donor and host. Burchardt[4] has studied the outcome of allograft transplantation in canine fibula and classified bone allograft incorporation into three distinct types.

Type I.—Repair was normal and had a clinically acceptable course. No fatigue

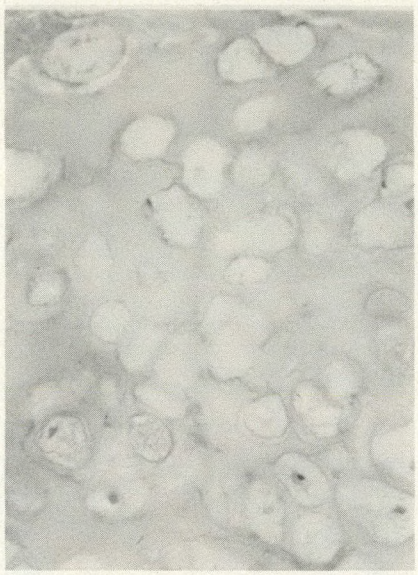

FIG. 5—Photomicrograph of tumour biopsy shows typical characteristics of low-grade chondrosarcoma (hematoxylin and eosin, original magnification × 400).

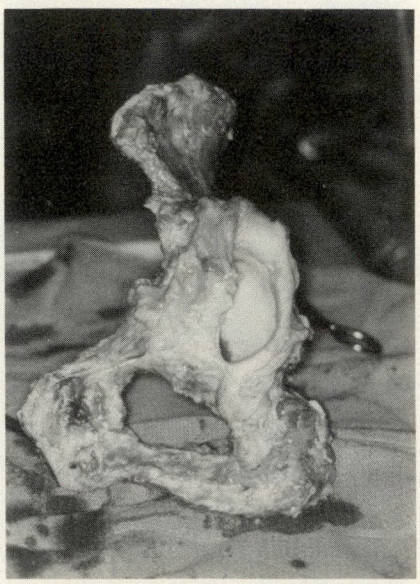

FIG. 6—Hemipelvic allograft.

Fig. 7a

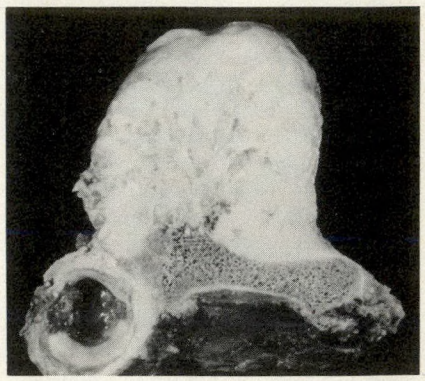

Fig. 7b

FIG. 7—(a) Resected specimen. (b) Specimen bisected to demonstrate huge chondrosarcoma.

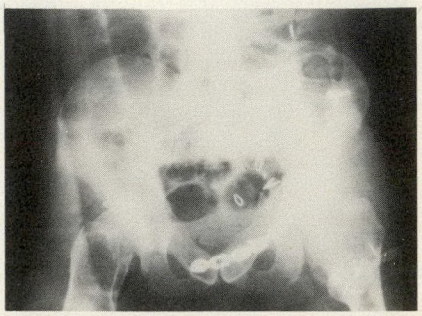

FIG. 8—Postoperative roentgenogram of transplanted allograft rigidly fixed with cancellous bone screws posteriorly and pelvic plate and screws anteriorly.

failures occurred and the graft was united to the host in 16 weeks. Radiologic remodelling and incorporation at 6 months were identical to those of autografts. The physical and biologic histomorphometric data and the corresponding microscopic data matched those of the autografts. This occurred in 20% of the animals, suggesting minimal or no significant immunologic differences between host and donor.

Type II.—Repair was slow and suggested greater genetic differences. These grafts were characterized by (a) an increased incidence of nonunion and delayed union, (b) unrepaired peripheral resorption with a decrease in graft size and (c) a substantial decrease in mechanical strength. These occurred in 60% of the animals and suggested a substantial immunologic difference but not great enough to cause complete resorption of the graft.

Type III.—Repair involved rapid and complete resorption of the graft with radiologic and histomorphometric data showing no repair and unrelenting resorption. This occurred in 20% of the animals.

To decrease the effects of genetic disparity between host and allograft, various graft modifications such as freeze-drying and deep freezing have been used and their effects on graft incorporation documented. These modifications were considered necessary by several researchers who showed that fresh allograft implanted against a strong histocompatibility barrier resulted in massive resorption of the implanted graft.[5–8]

Immunologic Aspects of Bone Allografts

Efforts to define bone allograft authenticity were begun in the 1950s by investigators such as Kreuz, Bonfiglio, Burwell and Chalmers. Their work was based on the histologic evaluation of the graft response, while others studied the patterns of skin-graft rejection after primary sensitization with bone. More recent work[9] has defined specific sources of antigenicity and means of decreasing the evoked immune responses. It is generally agreed from the above-mentioned studies that freezing greatly reduces bone allograft immunogenicity and that lyophilization (freeze-drying) further reduces the sensitizing properties. Fresh allografts have been found to be the most immunogenic.

Bone is composed of inorganic minerals, collagen, ground substance and a variety of cells. The only component without any immunogenic property is the inorganic minerals or the hydroxyapatite crystals.[3]

Collagen is a weak antigen but the importance of this is unknown. Various components of the ground substance, especially the proteoglycan subunits and link proteins have been shown to evoke an immune response in allogeneic and xenogeneic systems.[3]

By far the most potent immunogenic component of osteochondral allografts is the cell surface transplantation antigens.[3,9] These surface membrane glycoproteins are expressed under the major histocompatibility complex and are common to osteogenic and chondrogenic cells and also to fibroblastic, vascular, fatty, neuronal and hematopoietic cells.[3] Esses and Halloran,[10] using radiation chimeras, showed that the most potent source of immunogenic cells within bone allografts resides in the bone marrow. Czitrom and associates[11] concluded that these cells were neither T nor B lymphocytes but were of granulocyte lineage, resided in the bone marrow and initiated an immunologic reaction in vitro. To date, these cells have not been identified further and it is postulated that their removal would decrease the immunologic response to bone allografts.

The majority of the work in allograft immunology has been performed on animal models and little information is available on human osteochondral allograft immunology. Reports showing production of humoral antibodies in response to fresh osteochondral grafts[12] and the production of anti-HLA antibodies in recipients of freeze-dried bone allografts have been consistent. A recent report[13] cited the case of a 23-year-old Rh-negative woman who became sensitized after a frozen allograft of femoral heads had been used to fill in a defect in her distal tibia. The donors of the two femoral heads were Rh positive.

The biologic and clinical importance of immune responses to bone allografts remains unclear. It is true that fresh (unfrozen) osteochondral allografts evoke a demonstrable humoral and cell-mediated immune response and that these tissues undergo an unpredictable and usually unsatisfactory incorporation process,[3] although investigators have reported satisfactory clinical results using fresh osteochondral allografts.[14] They believe that fresh allografts induce blocking antibodies that diminish the immune response in vivo even though a demonstrable immune response is present in vitro.

The Bone Bank

In order to keep abreast of challenges in limb-preserving reconstructive surgery, a readily accessible bank of "biologic parts" must be available. For the past 12 years, large osteoarticular allografts and intercalary (interpositional) bone allografts have been prepared and banked.

It has been shown that tissue and organ allografts have served inadvertently as vectors for hepatitis, fatal slow viral diseases and even tumours that have metastasized and caused death. Recently, these fears have been amplified by the increasing awareness of acquired immune deficiency syndrome (AIDS). In order to avoid the transmission of disease from donor to recipient, strict criteria have been established for the selection of potential donors. In general, a review of the individual's medical history, circumstances of death, laboratory tests to detect venereal disease or hepatitis and an autopsy (in case of cadaver donors) form the bases of screening procedures. Potential donors are excluded if there is any suggestion of local or systemic evidence of infection. A history of hepatitis, malignant disease, diffuse connective tissue disorder, metabolic bone disease, serious systemic disorder of unknown etiology, chronic drug abuse, high-dose irradiation to the tissues being collected, or the presence of toxic substances that may be transferred in toxic amounts also exclude the donor. Once a donor has been secured, meticulous preparations for the retrieval of the specific tissue are required.[3]

Although it is possible to retrieve bone allografts in a nonsterile environment and later sterilize them by chemosterilization (ethylene oxide) or high-dose irradiation, most centres prefer retrieval in an operating room using sterile surgical techniques. Bone should be procured within 24 hours of the donor's death to reduce overgrowth of skin organisms. First, intracardiac blood, urine and pleural cultures should be obtained, as well as aerobic and anaerobic cultures of the bone allograft itself. Virtually any of the long bones, ribs or pelvis may be removed by subperiostal dissection followed by transection at various levels or disarticulation. Ligaments and capsular structures are left intact to aid in the subsequent reconstructive procedures.[3,15]

After the bones have been procured, proper preservation and storage are mandatory to retain their biologic potential. The biomechanical properties of allograft bone can be altered by the methods chosen for its preservation and storage. These effects are minimal with deep-freezing or low-level radiation. Freeze-drying markedly reduces the torsional and bending strength of bone allografts but does not deleteriously affect the compressive or tensile strength.[16] It has clearly been established that bone may be stored in a nonviable state with respect to cells and still function biologically, but cartilage must retain chondrocyte viability in order to support the specialized articular matrix required for a durable joint surface. At present, there is insufficient experimental or clinical data to demonstrate the clear superiority of any single approach to the preservation of all osteochondral grafts.[3]

Major bone allograft centres prefer to deep freeze their osteochondral tissues to temperatures of −70°C to −80°C. At these temperatures, enzymatic destruction is minimal and collagenase is inactive. Refrigeration at temperatures of −179°C with liquid nitrogen has been tried and may become the preferred approach, since at this temperature, molecular motion is minimal and tissue destruction is doubtful.[15] Two methods of preserving articular cartilage involve the immersion of the cartilaginous portion of the osteochondral allograft into 10% sterile glycerol or dimethylsulfoxide (DMSO) for cryoprotection of chondrocytes before freezing. Both agents seem to preserve approximately 40% of chondrocytes in a functional state,[17] although the precise number of chondrocytes required to maintain the articular matrix is unknown. Following this initial cryopreservation of the cartilage, the bone is placed in a −70°C freezer where it is stored until needed for clinical use.

The other popular method of storage is freeze-drying. The major advantage of this method is that bone can be stored indefinitely and it is particularly useful in a crushed form to fill cystic defects. The disadvantages are that viability of articular cartilage is destroyed and the strength of bone is altered as manifested by the brittle nature of the graft.[3,15]

Before thawing and subsequent implantation of the allograft, repeat cultures should be obtained and an antibiotic added to the thawing Ringer's solution. The bone graft should soak for approximately 1 hour.[15]

Clinical Applications

Bone allografts can be used in almost any part of the skeleton to fill, repair or replace diseased segments. The mode of use involves three categories: osteochondral allografts, intercalary allografts or prostheses coupled with bony allografts. The conditions for which bone allografts are used include simple bone grafts for nonunion of fractures, large allografts following tumour resections and for deficient bone stock during revision surgery for failed arthroplasty.

Gross and colleagues[14] have published numerous articles on the use of osteochondral allografts about the knee. In a series from 1971 to 1982, they reported on 110 osteochondral transplants for treating skeletal defects caused by degenerative, traumatic and neoplastic diseases of the knee. Seventy-eight small-fragment fresh allografts were transplanted to repair old osteochondral fractures of the tibial plateau for osteonecrosis or unicompartmental osteoarthritis. Thirty-two large-fragment grafts were performed following en bloc excision of bone tumours such as giant-cell tumour, osteogenic sarcoma, parosteal sarcoma, fibrosarcoma, angiosarcoma, ameloblastoma and chondrosarcoma. Their results "proved particularly rewarding in the old plateau fractures, for traumatic loss of bone and cartilage (osteonecrosis) and after en bloc excision of giant cell tumors".[14] Mankin and associates[1] have reported greater than 70% excellent or good results in patients who underwent bone allograft implantation for neoplastic disease. They also showed better results in patients with low-grade or benign tumours as well as resections and transplantations that did not involve a joint. The use of adjuvant chemotherapy following allograft implantation does not seem adversely to affect graft integration but does tend to increase postoperative complications.[18] The most devastating complication in allograft surgery is infection. Other complications include skin sloughs, allograft fracture, nonunion and graft resorption.

We thank Dr. C.A. Laurin for his constructive criticism and assistance in the preparation of this manuscript.

References

1. MANKIN HG, DOPPELT S, TOMFORD W: Clinical experience with allograft implantation. The first ten years. *Clin Orthop* 1983; 174: 69–86
2. BURWELL RG: *The Fate of Bone Grafts, Recent Advances in Orthopaedics*, Churchill, 1969: 115–207
3. FRIEDLAENDER GE, MANKIN HJ: Transplantation of osteochondral allografts. *Annu Rev Med* 1984; 35: 311–324
4. BURCHARDT H: The biology of bone graft repair. *Clin Orthop* 1983; 174: 28–42
5. HEIPLE KG, CHASE SW, HERNDON CH: A comparative study of the healing process following different types of bone transplantation. *J Bone Joint Surg [Am]* 1963; 45: 1593–1616
6. HERNDON CH, CHASE SW: Experimental studies in the transplantation of whole joints. *J Bone Joint Surg [Am]* 1952; 34: 564–578
7. CURTISS PJ JR, CHASE SW, HERNDON CH: Immunological factors in homogeneous bone transplantation. II. Histological studies. *J Bone Joint Surg [Am]* 1956; 38: 324–328
8. KREUZ FP, HYATT GW, TURNER TC, et al: The preservation and clinical use of freeze-dried bone. *J Bone Joint Surg [Am]* 1951; 33: 863–872
9. FRIEDLAENDER GE: Immune responses to osteochondral allografts. Current knowledge and future directions. *Clin Orthop* 1983; 174: 58–68
10. ESSES SI, HALLORAN PF: Donor marrow-derived cells as immunogens and targets for the immune response to bone and skin allografts. *Transplantation* 1983; 35: 169–174
11. CZITROM AA, AXELROD T, FERNANDES B: Antigen presenting cells and bone allotransplantation. *Clin Orthop* 1985; 197: 27–31
12. LANGER F, GROSS AE, WEST M, et al: The immunogenicity of allograft knee joint transplants. *Clin Orthop* 1978; 132: 155–162
13. JOHNSON CA, BROWN BA, LASKY LC: Rh immunization caused by osseous allograft (C). *N Engl J Med* 1985; 312: 121–122
14. GROSS AE, MCKEE NH, PRITZKER KP, et al: Reconstruction of skeletal deficits at the knee. A comprehensive osteochondral transplant program. *Clin Orthop* 1983; 174: 96–106
15. TOMFORD WW, DOPPELT SH, MANKIN HJ, et al: 1983 bone bank procedures. *Ibid:* 15–21
16. PELKER RR, FRIEDLAENDER GE, MARKHAM TC: Biomechanical properties of bone allografts. *Ibid:* 54–57
17. TOMFORD WW, MANKIN HJ: Investigational approaches to articular cartilage preservation. *Ibid:* 22–27
18. DICK HM, MALININ TI, MNAYMNEH WA: Massive allograft implantation following radical resection of high-grade tumors requiring adjuvant chemotherapy treatment. *Clin Orthop* 1985; 197: 88–95

24 hours following surgery increases the possibility of adverse reactions but, in the majority of surgical procedures, does not reduce the incidence of subsequent infection.

If signs of postsurgical infection should appear, specimens for culture should be obtained for identification of the causative organism(s) so that appropriate therapy may be instituted.

CONTRAINDICATIONS

MEFOXIN* is contraindicated in persons who have shown hypersensitivity to cefoxitin or to the cephalosporin group of antibiotics.

WARNINGS

Before therapy with MEFOXIN* is instituted, careful inquiry should be made to determine whether the patient has had previous hypersensitivity reactions to MEFOXIN*, cephalosporins, penicillins or other drugs. MEFOXIN* should be given with caution to penicillin-sensitive patients.

There is some clinical and laboratory evidence of partial cross-allergenicity between cephamycins and the other beta-lactam antibiotics, penicillins and cephalosporins. Severe reactions (including anaphylaxis) have been reported with most beta-lactam antibiotics.

Pseudomembranous colitis has been reported with virtually all antibiotics. This colitis can range from mild to life threatening in severity. Antibiotics should therefore be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis. It is important to consider a diagnosis of pseudomembranous colitis in patients who develop diarrhea in association with antibiotic use. While studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis, other causes should also be considered.

Any patient who has demonstrated some form of allergy, particularly to drugs, should receive antibiotics including MEFOXIN* with caution.

If an allergic reaction to MEFOXIN* occurs, administration of the drug should be discontinued. Serious hypersensitivity reactions may require treatment with epinephrine and other emergency measures.

PRECAUTIONS

The total daily dosage should be reduced when MEFOXIN* is administered to patients with transient or persistent reduction of urinary output due to renal insufficiency (see DOSAGE AND ADMINISTRATION) because high and prolonged serum antibiotic concentrations can occur from usual doses.

In patients treated with MEFOXIN* a false-positive reaction to glucose in the urine may occur with Benedict's or Fehling's solutions but not with the use of specific glucose oxidase methods.

Using the Jaffe Method, falsely high creatinine values in serum may occur if serum concentrations of cefoxitin exceed 100 μg/mL. Serum samples from patients treated with MEFOXIN* should not be analyzed for creatinine if withdrawn within two hours of drug administration.

Increased nephrotoxicity has been reported following concomitant administration of cephalosporins and aminoglycoside antibiotics.

The safety of MEFOXIN* in the treatment of infections during pregnancy has not been established. If the administration of MEFOXIN* to pregnant patients is considered necessary, its use requires that the anticipated benefits be weighed against possible hazards to the fetus. Reproductive and teratogenic studies have been performed in mice and rats and have revealed no evidence of impaired fertility or harm to the fetus due to MEFOXIN*.

Cefoxitin has been observed in the milk of nursing mothers receiving the drug.

Prolonged use of MEFOXIN* may result in the overgrowth of non-susceptible organisms. Repeated evaluation of the patient's condition is essential and if super-infection occurs during therapy, appropriate measures should be taken. Should an organism become resistant during antibiotic therapy, another antibiotic should be substituted.

In children 3 months of age or older, higher doses of MEFOXIN* (100 mg/kg/day and above) have been associated with an increased incidence of eosinophilia and elevated SGOT.

ADVERSE REACTIONS

MEFOXIN* is generally well tolerated. Adverse reactions rarely required cessation of treatment and usually have been mild and transient.

Local Reactions
Thrombophlebitis has occurred with intravenous administration. Some degree of pain and tenderness is usually experienced after intramuscular injections using water. Induration has occasionally been reported.

Allergic
Maculopapular rash, urticaria, pruritus, eosinophilia, fever and other allergic reactions have been noted.

Gastrointestinal
Symptoms of pseudomembranous colitis can appear during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Blood
Transient eosinophilia, leukopenia, neutropenia, hemolytic anemia, and thrombocytopenia have been reported. Some individuals, particularly those with azotemia, may develop positive direct Coombs tests during therapy with MEFOXIN*.

Liver Function
Transient elevations in SGOT, SGPT, serum LDH, and serum alkaline phosphatase have been reported.

Kidney
Elevations in serum creatinine and/or blood urea nitrogen levels have been observed. As with the cephalosporins, acute renal failure has been reported rarely. The role of MEFOXIN* in changes in renal function tests is difficult to assess, since factors predisposing to prerenal azotemia or to impaired renal function have often been present.

TREATMENT OF OVERDOSE

Other than general supportive treatment, no specific antidote is known. MEFOXIN* can be eliminated by dialysis in patients with renal insufficiency.

DOSAGE AND ADMINISTRATION

MEFOXIN* may be administered intravenously or intramuscularly when required. (See complete monograph for full details on ADMINISTRATION and RECONSTITUTION.)

TREATMENT DOSAGE

Adults

The usual adult dosage is 1 g or 2 g of MEFOXIN* every 6 to 8 hours. Dosage and route of administration should be determined by severity of infection, susceptibility of the causative organisms, and condition of the patient. The usual adult dosages are shown in the Table below.

Usual Adult Dosage

Type of infection	Daily Dosage	Frequency and Route
Uncomplicated forms* of infections such as pneumonia, urinary tract infection, soft tissue infection	3-4 g	1 g every 6-8 h I.V. or I.M.
Moderately severe or severe infections	6-8 g	1 g every 4 h or 2 g every 6-8 h I.V.
Infections commonly needing antibiotics in higher dosage (e.g. gas gangrene)	12 g	2 g every 4 h or 3 g every 6 h I.V.

*Including patients in whom bacteremia is absent or unlikely

Therapy may be started while awaiting the results of susceptibility testing.

Antibiotic therapy for group A beta-hemolytic streptococcal infections should be maintained for at least 10 days to guard against the risk of rheumatic fever or glomerulonephritis. In staphylococcal and other infections involving a collection of pus, surgical drainage should be carried out where indicated.

Adults with Impaired Renal Function

MEFOXIN* may be used in patients with reduced renal function but a reduced dosage should be employed and it is advisable to monitor serum levels in patients with severe impairment.

In adults with renal insufficiency, an initial loading dose of 1 g to 2 g should be given. After a loading dose, the following recommendations for **maintenance dosage** may be used as a guide:

RENAL FUNCTION	CREATININE CLEARANCE mL/min	DOSE	FREQUENCY
Mild impairment	50-30	1-2 g	every 8-12 h
Moderate impairment	29-10	1-2 g	every 12-24 h
Severe impairment	9-5	0.5-1 g	every 12-24 h
Essentially no function	<5	0.5-1 g	every 24-48 h

In the patient undergoing hemodialysis, the loading dose of 1-2 g should be given after each hemodialysis, and the maintenance dose should be given as indicated in the Table above.

Neonates (Including Premature Infants), Infants and Children (See WARNINGS for Neonates under ADMINISTRATION in the complete monograph.)

Premature Infants with Body Weights Above 1500 g	20-40 mg/kg every 12 h I.V.
Neonates	
0-1 week of age	20-40 mg/kg every 12 h I.V.
1-4 weeks of age	20-40 mg/kg every 8 h I.V.
Infants	
1 month to 2 years of age	20-40 mg/kg every 6 h or every 8 h I.M. or I.V.
Children	20-40 mg/kg every 6 h or every 8 h I.M. or I.V.

In severe infections, the total daily dosage in infants and children may be increased to 200 mg/kg, but not to exceed 12 g per day.

MEFOXIN* is not recommended for the therapy of meningitis. If meningitis is suspected, an appropriate antibiotic should be used.

At present there is insufficient data to recommend a specific dosage for children with impaired renal function. However, if the administration of MEFOXIN* is deemed to be essential the dosage should be modified consistent with the recommendations for adults (see Table above).

PROPHYLACTIC USE

For prophylactic use, a three-dose regimen of MEFOXIN* is recommended as follows:

Vaginal or abdominal hysterectomy and abdominal surgery

2 g administered intramuscularly or intravenously just prior to surgery (approximately one-half to one hour before initial incision).

The second and third 2 g doses should be administered at 2-6 hour intervals after the initial dose.

Cesarean Section

The first dose of 2 g should be administered intravenously as soon as the umbilical cord has been clamped. The second and third 2 g doses should be given intravenously or intramuscularly four hours and eight hours after the first dose.

AVAILABILITY

MEFOXIN* (sterile cefoxitin sodium, Frosst Std.) is supplied as sterile powder in boxes of 10 vials:

No. 3356 1 g cefoxitin as sodium salt
No. 3357 2 g cefoxitin as sodium salt

Storage
MEFOXIN* in the dry state should be stored below 30° C.

PRODUCT MONOGRAPH AVAILABLE ON REQUEST

421-a,11,84

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B. LAMBERT, MD;* M. LESSARD, MD†

Massive Ovarian Edema in a Twin Pregnancy

A case of idiopathic ovarian edema in a 26-year-old woman with a twin pregnancy is reported. The clinical evolution of the condition was benign in spite of acute episodes of left iliac pain. In diagnosis, theca-lutein cysts had to be differentiated from other ovarian enlargements. The authors advise conservative surgery for such a condition.

Les auteurs rapportent un cas d'oedème ovarian idiopathique durant une grossesse gémellaire. L'évolution clinique fut bénigne en dépit des douleurs à la fosse iliaque gauche. Il faut faire le diagnostic différentiel des kystes de la thèque lutéinisée et des autres kystes ovariens. La chirurgie conservatrice est de mise.

Massive ovarian edema was reported in 1969 by Kalstone and associates.[1] In 1984, Young and Scully[2] added 11 patients to the 40 already described in the literature and referred to 1 more reported by Fukuda. Recently VanWingen and colleagues[3] added a case of bilateral massive ovarian edema in a nulligravid patient. To date there have been only three cases of ovarian edema associated with pregnancy. Our case is the 54th reported and is unique for its association with a twin pregnancy.

Case Report

A 26-year-old woman was admitted at 12 weeks' gestation for acute pelvic pain radiating to the right lumbar region. She did not have any pollakiuria, dysuria, hematuria or vomiting. She had been under observation for oligomenorrhea. Ultrasonography confirmed a 12 weeks' twin pregnancy with a 9 cm right ovarian cyst. The pain persisted in spite of analgesics. A diagnosis of ovarian torsion was made and a laparotomy performed. A 16-cm gravid uterus was noted with a right ovarian mass measuring 12 × 15 cm bulging in the right hypochondrium. There was evidence of cyanosis, hemorrhage and necrosis. The right tube was involved in the process so a right adnexectomy was performed. The left ovary and tube were normal. The pathological diagnosis was hemorrhagic corpus luteum cyst of the right ovary. The postoperative recovery was uncomplicated.

The patient was readmitted at 27 weeks' gestation for recurrent, progressive, left iliac pain, radiating to the left lumbar region. B-scanning revealed a left ovarian cyst measuring 7 × 3.4 cm. The clinical impression was semitorsion of the ovary by a large corpus luteal cyst or a theca-lutein cyst. After 3 days of observation, the woman's condition had improved and she was discharged, although subsequent sonograms showed that the cyst was still present. She was delivered at 33 weeks' gestation of two diamniotic dichorionic girls. The postpartum sonogram showed a persistent left ovarian cyst measuring 9.7 × 5 cm. The cyst was resected but the ovary preserved (Fig. 1), and a left tubal ligation was performed. Recovery was smooth and postoperative sonography 5 weeks later gave normal results.

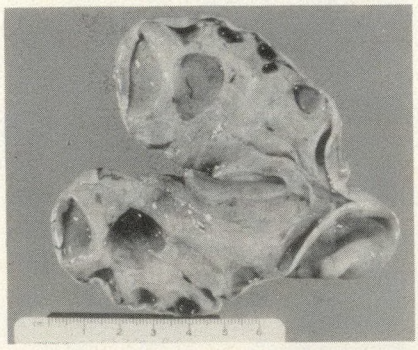

FIG. 1—Resected ovary.

The left ovary measured 11 × 5 × 2 cm and weighed 52 g. The surface was smooth and white. On section, multiple cystic cavities, measuring from 0.8 cm to 2.5 cm, were noted (Fig. 1). They contained a yellow liquid which was hemorrhagic in some areas. Signs of tubal hemorrhagic necrosis were also present. On microscopic examination there was a normal superficial cortical zone overlying multiple follicular cysts. The largest cyst was lined by luteinized cells. The stroma was markedly edematous with lymphatic distension. There were few isolated luteinized cells and no stromal hyperplasia.

Discussion

Massive ovarian edema is a tumour-like condition defined as marked enlargement of one or both ovaries by an accumulation of edema fluid in the stroma separating normal follicular structures. In some cases, the stroma contains lutein cells and the patient is virilized. The treatment must be conservative.

Young and Scully[2] in their recent comprehensive review found that the typical patients are young women with an aver-

Table I—Reported Cases of Massive Ovarian Edema in Pregnancy

Report	Patient age, yr	Onset of symptoms, trimester	Operative findings		Follow-up
			Size, cm	Ovary	
Gustafson and associates, 1954[4]	22	2	9	Right	–
Chervenak and associates, 1980[5]	22	3	28	Right	Premature delivery 2 d postop
Young and Scully, 1984[2]	16	1	8	Right	–
Present case	26	3	11	Left	Twin pregnancy with premature delivery at 33 wk

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age age of 22 years (range from 6 to 33 years). The clinical picture is variable. Most often the patient presents with abdominal pain, sometimes mimicking acute appendicitis. There can also be asymptomatic abdominal swelling and menstrual irregularities. The condition is usually unilateral, although in seven reported cases it was bilateral. In approximately 59% of cases there was a partial or complete torsion of the ovarian pedicle.

Grossly, the ovary is enlarged, ranging in dimension from 5.5 to 35 cm. The surface is white, smooth and shiny. On section, the stroma seems edematous and there are a few cystic cavities. Microscopically, multiple follicular cysts, edematous stroma with dissociated spindle cells and occasionally luteinized cells are seen.

Two theories are proposed to explain the lesion: first, torsion of the ovary, either partial or complete, with subsequent enlargement of the ovary and edema and, second, stromal proliferation that may predispose to ovarian torsion with subsequent edema. These simple mechanisms could explain many cases of massive ovarian edema. Unfortunately in 11 reported cases (21%) the authors did not find ovarian torsion during the surgical exploration.

The patient described in this report was a typical case. During the first laparotomy, the left ovary was normal although the right ovary contained a hemorrhagic corpus luteum cyst. The sudden onset of abdominal pain favours the first theory. It is the fourth reported case associated with pregnancy which in this case is unique in being a twin pregnancy (Table I[2,4,5]).

In summary, we cannot predict the influence of such a lesion on the pregnancy. In the presence of a twin pregnancy, the possibility of theca-lutein cysts[6] cannot be ruled out. The surgical attitude must be conservative, based on peroperative pathological examination.

References

1. KALSTONE CE, JAFFE RB, ABELL MR: Massive edema of the ovary simulating fibroma. *Obstet Gynecol* 1969; 34: 564–571

2. YOUNG RH, SCULLY RE: Fibromatosis and massive edema of the ovary, possibly related entities: a report of 14 cases of fibromatosis and 11 cases of massive edema. *Int J Gynecol Pathol* 1984; 3: 153–178

3. VANWINGEN T, UPTON RT, CLOHERTY MG, et al: Bilateral massive ovarian edema. A case report. *J Reprod Med* 1984; 29: 875–877

4. GUSTAFSON FW, GARDINER SH, STOUT FE: Ovarian tumors complicating pregnancy. A review of 45 surgically proved cases. *Am J Obstet Gynecol* 1954; 67: 1210–1223

5. CHERVENAK FA, CASTADOT MJ, WIEDERMAN J, et al: Massive ovarian edema: review of world literature and report of two cases. *Obstet Gynecol Surv* 1980; 35: 677–684

6. LAMBERT B, DEBRUX J: Theca lutein cysts of pregnancy without mole or chorio-epithelioma. *Obstet Gynecol* 1963; 22: 643–647

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Manuscripts of original articles and other contributions, including a limited number of case reports, should be submitted, in triplicate, *in English or French, with a covering letter requesting consideration for publication. Authors must also include a statement indicating that the manuscript is not under consideration by any other journal and has not been published previously. The manuscript should be typed on one side of plain paper, double spaced with wide margins. Measurements should be expressed according to the* Système international d'unités.

Illustrations (e.g., photographs of clinical material, roentgenograms, photomicrographs, graphs and diagrams), in triplicate, *should be in the form of glossy, unmounted and untrimmed prints, not larger than 20 × 25 cm. A legend must be supplied for each; the legend(s) should be typed on a page separate from the text of the article. For a roentgenogram submit a print rather than the original; for a photomicrograph include details of the stain used and the magnification in the legend. Lettering identifying parts of the illustration should be large enough to remain visible when the illustration is reduced in size for publication. A patient must not be recognizable unless written consent has been obtained; otherwise facial features may require blocking. Colour work can be published only at the author's expense. If an illustration is taken from a source other than the author's, letters of permission to reproduce must be obtained from the original publisher and author.*

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BRIAN J. MILLER, MB, FRCSC;* GORDON M. WYANT, MD, FRCPC†

The Epidural Opioid Internalized System

For several years opioids have been given epidurally with success to control chronic intractable pain. The authors report their 2-year experience with internalization of the epidural catheter and injection port in 18 patients at University Hospital, Saskatoon. In all, 21 devices were used. All patients had metastatic cancer; 16 are now dead. The need for other medications was eliminated with 9 and reduced with 12 devices. In the patients who died, the devices were effective for 82% of their remaining lifespan. They were able to spend 46% of this time at home. Blockage of the epidural end of the device was the most common mechanical problem, followed by leakage from the port. One patient suffered meningitis after 11 days as a result of subarachnoid placement of the catheter but responded to removal of the device and antibiotic therapy. The authors have been impressed with the excellent pain relief afforded to many of these patients, and their ability to enjoy life free of the sedative effects of other methods of narcotic administration.

Depuis plusieurs années, les opiacés sont administrés avec succès par voie épidurale pour maîtriser la douleur chronique réfractaire. Les auteurs décrivent leur utilisation pendant 2 ans d'un cathéter épidural à demeure muni d'un embout d'injection, chez 18 patients de l'Hôpital universitaire de Saskatoon. En tout, 21 appareils ont été utilisés. Tous les patients avaient des métastases cancéreuses; 16 sont maintenant décédés. Neuf appareils ont permis de supprimer les médications supplémentaires, et les 12 autres en ont réduit le besoin. Pour les patients qui sont décédés, les appareils se sont révélés efficaces pendant 82% du temps qui leur restait à vivre. Ils ont pu ainsi passer 46% de ce temps à domicile. L'obstruction de l'extrémité épidurale de l'instrument fut le problème mécanique le plus fréquent; venait ensuite, la fuite de l'embout. Un patient a souffert de méningite au bout de 11 jours, des suites de la mise en place du cathéter dans l'espace sous-arachnoïdien; le patient a réagi favorablement au retrait de l'instrument et à un traitement antibiotique. Les auteurs ont été favorablement impressionnés par l'excellent soulagement de la douleur dont bénéficièrent plusieurs patients, de même que de la possibilité qui leur était offerte de jouir de la vie sans les effets sédatifs que produisent les autres méthodes d'administration des substances stupéfiantes.

The practice of injecting opioids into the epidural space has come into vogue as an effective method for alleviating intractable pain in thoracic, abdominal and lower extremity areas, with little systemic effect. This paper describes our experience starting in February 1984 with an internalized system for the long-term delivery of morphine to the epidural space in 18 patients, suffering from malignant disease, with painful metastases.

Patients, Materials and Methods

The 18 patients (9 men, 9 women) ranged in age from 25 to 70 years (mean 55 years); 10 were 60 years or older. Many primary tumours were represented, the commonest being bronchogenic carci-

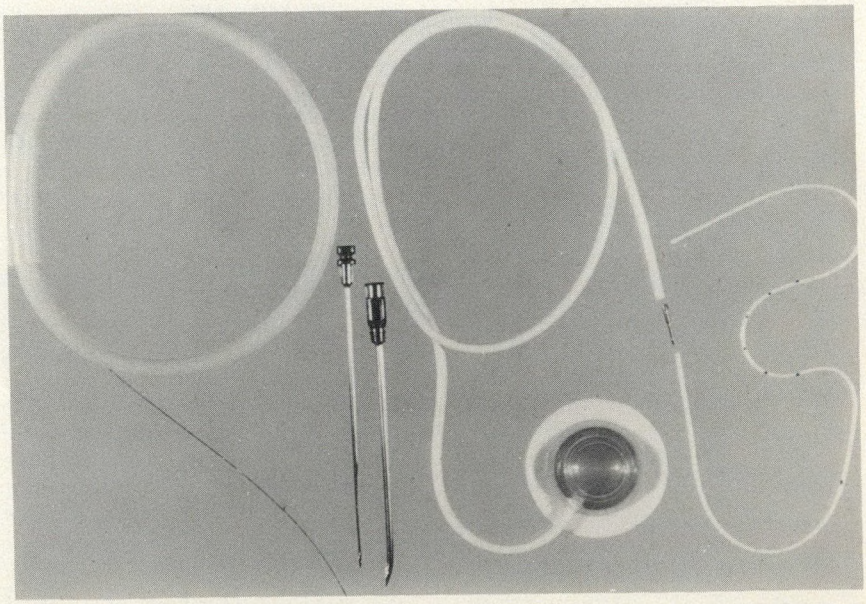

FIG. 1—Components of Holter-Hausner device: port and its tubing, epidural tubing, metal connector for two tubes and no. 14 Tuohy needle.

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noma in four patients. The metastases were located in the thoracolumbar spine and pelvis in most patients. One patient had painful brachial plexus metastases from a melanoma primary. Preoperative pain medications included parenterallly administered morphine in eight patients, up to 1400 mg/d and a variety of other narcotics (meperidine, codeine, anileridine and levorphanol).

In the first three patients an Omaya device (McGraw Supply Ltd., Mississauga, Ont.) was placed initially. This system was unsatisfactory because of leaks and lack of a steel back to the injection port. The Holter-Hausner (Holter-Hausner International, Inc.; Canada Microsurgical Ltd., Burlington, Ont.) implantable epidural opioid system was used in the other 15 patients. It consists of a 1-ml Silastic-topped reservoir with a steel back and a broad Silastic flange to be sutured to the underlying fascia. Silastic tubing leads away from the port and is connected to the free end of the Silastic epidural catheter by a small metal adapter (Fig. 1). A no. 14 Tuohy needle is required to introduce the epidural catheter. A wire stylet is provided to stiffen the epidural catheter for insertion into the epidural space through the Tuohy needle. The port can be pierced with a 25-gauge needle through the centre several hundred times before leakage becomes substantial, because of the thickness of the Silastic in this area.

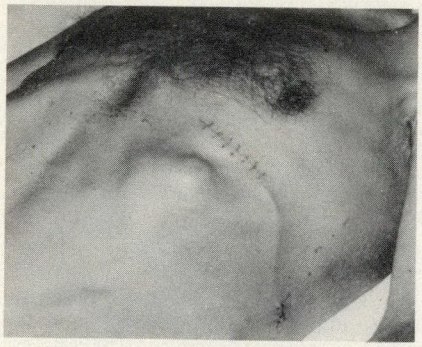

FIG. 2—In thin patients, injection port can be seen clearly overlying left inframammary ribs with port tubing leading away posteriorly towards spine.

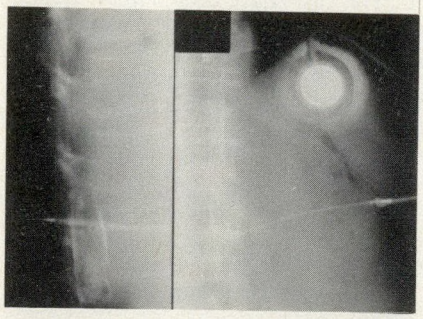

FIG. 3—Epidurogram demonstrates extravasation of dye around port although some does reach epidural space.

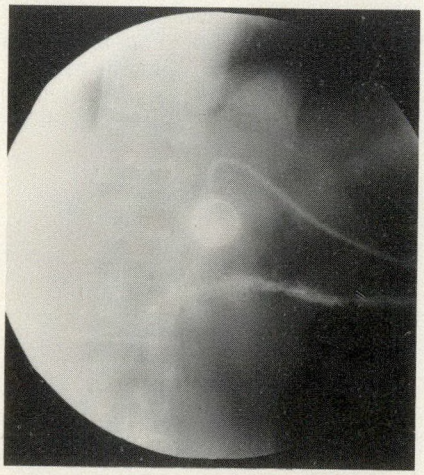

FIG. 4—Dye study shows normal port, but there is extravasation of dye close to metal connector and none reaches epidural space, where tubing is blocked.

We used a general anesthetic to place the device. The patient is positioned on the right side and the external epidural catheter is removed. The epidural tube is inserted first through a small skin incision and its position checked by injecting a small amount of diatrizoate radiopaque dye. Without changing the patient's position, the epidural tube is tunnelled subcutaneously to the left of the spine for a few centimetres and brought out through a short transit incision. A 5-cm transverse incision is then made just below the left inframammary crease over the ribs in that area, and a subcutaneous pocket is dissected below it for the port. The port is placed and anchored to the underlying fascia by nylon sutures through the flange. The port tubing is then led laterally through a subcutaneous tunnel and united with the epidural tubing over the metal connector. All incisions are then closed with nylon sutures. Morphine may be injected immediately (Fig. 2), unless this was done before the external catheter was removed.

One of us (G.M.W.) was responsible for selecting the patients and siting the epidural catheters. Three staff surgeons at University Hospital, Saskatoon (including B.J.M.) were responsible for tunnelling the catheters and siting the ports. The selection process involved an assessment of the expected lifespan. Patients who were not expected to survive more than a few weeks were excluded, as were those who were unlikely to be discharged from hospital. Other methods were adopted for pain control in these cases. In potentially suitable patients, a trial of epidural morphine pain control was undertaken by means of a standard external epidural catheter. The duration of these trials was 2 to 27 days (mean 10 days). If the control so achieved was worthwhile, the patient was considered for internalization. Major sepsis elsewhere in the body was a contraindication to implantation. Patients were taught to self-inject before discharge, if possible.

If there was significant loss of pain control after implantation, we attempted to diagnose the fault in the system by a dye study, injecting MD-60 dye into the port under radiologic control, looking for extravasation (Fig. 3) or blockage (Fig. 4).

Results

Three of the patients required replacement of devices for a total of 21 devices implanted.

Operating time for insertion of the internalized epidural port ranged from 30 minutes to 95 minutes (mean 50 minutes). Two out of the three Omaya devices required replacement for port leakage. The spinal insertion level for the epidural catheter was most commonly at L2–3 (nine patients), followed by L3–4 (five patients). The highest level of insertion was at T3–4, and the catheter was advanced into the cervical epidural space for control of brachial plexus pain. Morphine dosage into the epidural port ranged between 15 mg and 25 mg/d in two or three divided doses.

The effective device time ranged from 3 days to 480 days. Seven devices were effective for less than 30 days, 11 devices for 30 to 90 days and 3 for more than 90 days. Within this time there was complete relief of pain with no need for any other pain medications with 9 devices and partial effectiveness with the need for additional analgesics in 12. The range of survival of the 16 patients who died was from 3 days to 590 days. Four patients survived less than 30 days from the time of implantation, six survived between 30 and 90 days and six survived for more than 90 days after implantation.

The effective device time to patient survival was found to be 82% overall. When the four patients who survived less than 30 days are excluded, the effective device time to patient survival was 78%. A review of the number of days that patients spent at home after implantation revealed that the proportion of discharge days to total survival days was 46%. Five patients died without leaving hospital.

Complications

Technical problems with the reservoir end of the system included two port leaks, occurring at 90 and 200 days respectively, and one tubing leak that occurred at 14 days. These all happened as a result of gashes by misplaced 25-gauge needles. Mechanical problems at the epidural end of the system included slippage from the epidural space in two cases at 1 day and 90 days, and blockage in five cases between 14 days and 90 days.

Subarachnoid placement of the epidural catheter occurred once and was recognized in the operating room. However, this patient suffered meningitis after 11 days and the whole device had to be removed. He recovered with antibiotic therapy. One patient died of hypercalcemia and coagulopathy secondary to massive metastases from carcinoma of the breast only 3 days after implantation. Two dural punctures were made inadvertently during insertion but there were no sequelae. Five patients experienced pain postoperatively at the time of morphine top-up. This was partially alleviated in three by temporary predosing with lidocaine into the port and in one patient by withdrawal of the catheter by 1 cm; in the other patient the pain resolved spontaneously. In one patient the device was underutilized at home after discharge because of uncooperative relatives.

Discussion

The problem of pain control in the chronically ill patient is common to every clinician. Often palliation is unsatisfactory. These patients may spend their days, which in the case of metastatic disease may be limited to weeks or months, unable to enjoy their homes and families, either because of persisting intractable pain or from oversedation with narcotic analgesics. Many measures have been tried, including drug therapy with oral preparations of the narcotics, as well as mixtures of narcotics, sedatives and antiemetics. Operative procedures such as nerve injection and section, cordotomy and thalamotomy are occasionally advocated.

Epidural opioid injection has been used for intractable pain for several years.[1] Coombs and associates[2] were one of the first groups to report on epidural port implantation techniques and efficacy. This route of narcotic administration has many advantages. The dose required for pain control is lower than that needed for systemic routes. There is no loss of normal sensory or motor function and little effect on the central nervous system. Provided the relevant areas of the spinal cord are within reach of the morphine in its diluent vehicle in the epidural space, the need for other analgesics can be eliminated or substantially reduced.

The major disadvantage has been the need for a standard catheter to the epidural space with an external component to allow repeated injection of morphine. The external injection port is inconvenient for the patient, who might be bedridden, and after 2 weeks or so the epidural space is susceptible to infection even with the best of attention to the dressings. Implantation of an injection port and Silastic tubing obviates these problems. The procedure is short and, in our experience, has been well tolerated even by very ill patients. The use of various techniques for insertion under local anesthesia has been well described.[3] We have found that a short general anesthetic is preferable in these patients, who often have painful metastases in the spine and ribs, and cannot maintain the right-side-down position on a hard table for long. Delivery of morphine by the port can start immediately after placement, and our objective is to have the patients familiar with self-injection before they leave hospital a few days later.

The morphine sulfate used differs from the usual parenteral solution of morphine only in that it does not contain preservative; other opioids, suitably diluted, may be used. The usual dose of morphine is 4 to 6 mg two to three times daily; occasionally up to 8 mg may be needed. The site of action of the morphine appears to be at the opiate μ receptors in the dorsal horn of the spinal cord. The morphine diffuses across the dura and subarachnoid space to gain entry to this area.[4] Each dose of morphine is made up in 10 ml of normal saline to achieve adequate dispersal through the epidural space.

Our patients underwent a trial of an external epidural system before implantation to ensure that pain relief would be worthwhile. This probably accounted for the lack of complete failures in our series. We attempted to select patients who had a reasonable life expectancy (more than a few weeks) but failed to achieve this in 4 of the 18 patients. In several patients, a dye study was utilized effectively to identify the cause of lost analgesic effect. Where there was obvious swelling of the subcutaneous pocket around the port at the time of injection, implying a leak at the port, the area was occasionally explored under local analgesia on the ward to confirm the leak. However, frequently the leak was secondary to blockage elsewhere in the system, and often the whole device had to be changed. Exploration of the metal connector under local anesthetic without x-ray films is an alternative to define which half of the system is faulty.

We have not yet used this device in any patient with benign disease. The published results for this so far have not been encouraging;[4] such patients appear to respond in a less predictable and satisfactory manner to continuous intraspinal narcotic analgesia. Coombs and colleagues[4] in their series of five such patients, suggested that the possibility of reward for pain may distort the patient's perception of his disability. Continuous infusion systems, which we have not used, have also to be considered for these patients as their survival is likely to be so long that repeated injections to a subcutaneous port would become unfeasible.

In our series we noted that the dose of epidural analgesic required for optimal pain relief gradually increased. This "tolerance" has been addressed in the literature.[4] Speculation as to the cause varies and includes progressive dural fibrosis as a reaction to the presence of the catheter, true spinal opiate tolerance and increasing pain from advancing malignant disease. We have encountered two instances in which patients became suddenly totally resistant to epidural morphine, but after 2 or 3 days of epidural fentanyl citrate (50 μg) they regained their response to morphine. Other complications, occasionally seen, include nausea, which is treated with standard antiemetics, urinary retention, which yields to parasympathomimetics such as bethanechol, and pruritis, which can be controlled with naloxone without losing the analgesic effect of the opioid.

In patients such as those in our series with advanced malignant disease, the issue of respiratory depression in response to epidural narcotic has not been a problem, perhaps because previous exposure to systemic narcotic analgesics appears to provide protection against this complication.[5]

In conclusion, our experience is derived from a relatively small uncontrolled series. Our cases have been notably free of serious side effects and complications, except for one patient who had meningitis. The Pain Management Service at University Hospital, in association with the Saskatoon Cancer Clinic, treats a large number of cancer patients, many with intractable pain, yet only 18 patients have been submitted to epidural implantation. This is an expression of rigorous patient selection, which is essential if good results are to be achieved. The results reported are particularly gratifying since all other avenues of pain relief had proven unsatisfactory for various reasons. The 46% post-implant discharge time is hard to assess without a control, but we have been impressed with the effectiveness of this technique both as a means of providing good palliation and allowing patients to return home to their families without the sedative effects of other methods of narcotic administration.

References

1. BROMAGE PR, CAMPORESI E, CHESTNUT D: Epidural narcotics for postoperative analgesia. *Anesth Analg* 1980; 59: 473–480
2. COOMBS DW, SAUNDERS RL, GAYLOR M, et al: Epidural narcotic infusion reservoir: implantation technique and efficacy. *Anesthesiology* 1982; 56: 469–473
3. CHERRY DA, GOURLAY GK, COUSINS MJ, et al: A technique for the insertion of an implantable portal system for the long-term epidural administration of opioids in the treatment of cancer pain. *Anaesth Intensive Care* 1985; 13: 145–152
4. COOMBS DW, SAUNDERS RL, GAYLOR MS, et al: Relief of continuous chronic pain by intraspinal narcotics infusion via an implanted reservoir. *JAMA* 1983; 250: 2336–2339
5. ZENZ M, SCHAPPLER-SCHEELE B, NEUHAUS R, et al: Long-term peridural morphine analgesia in cancer pain (C). *Lancet* 1981; 1: 91

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Transjugular Intrahepatic Portosystemic Shunt: a Nonoperative Approach to Life-Threatening Variceal Bleeding

Portosystemic venous shunts may be created nonoperatively with a Grüntzig balloon dilatation catheter using the transjugular route. The authors achieved technical success with this shunt in 15 of 20 patients with life-threatening gastrointestinal bleeding from variceal hemorrhage. All patients but one were considered at high risk for surgery because of end-stage liver disease; the exception was a patient in whom two previous operative portosystemic shunts had failed. An average decrease of 5.9 mm Hg in portal vein pressure was measured in 11 patients for whom sequential pressures could be obtained. Two patients survived longer than 12 months without subsequent operative procedures, and the shunt helped temporize in three other patients who later underwent operation. Nine patients with successful shunts died within 30 days of the procedure, comparing favourably with reported operative death rates of 40% to 80% in emergency shunt procedures. Follow-up portal venograms demonstrated shunt patency in six of nine patients, in one after 8 months. Tract patency was determined in four of seven patients on whom autopsy was performed, up to 6 months after the transjugular intrahepatic portosystemic shunt was created.

Une anastomose porto-cave peut être créée de manière non chirurgicale à l'aide d'un cathéter à ballonnet de dilatation Grüntzig. Les auteurs ont obtenu avec ce type d'anastomose, un succès technique dans 15 cas sur 20 d'hémorragies gastro-intestinales variqueuses menaçant la survie. Tous les patients sauf un, étaient considérés à risque opératoire élevé du fait d'une affection hépatique en phase terminale; un patient chez qui deux tentatives chirurgicales d'anastomoses porto-caves avaient échoué, constituait l'exception. Une diminution moyenne de 5.9 mm Hg de la pression de la veine porte a été mesurée chez 11 patients pour qui des mesures séquentielles de la pression ont pu être obtenues. Deux patients ont survécu pendant plus de 12 mois sans nécessiter d'interventions chirurgicales subséquentes. De plus, l'anastomose a permis de temporiser chez trois autres patients qui subirent plus tard une opération. Neuf patients chez qui l'anastomose fonctionna moururent dans les 30 jours qui suivirent l'intervention; ceci se compare favorablement à la mortalité opératoire de 40% à 80% rapportée dans les cas d'anastomose urgente. Des phlébogrammes portes de surveillance ont démontré la perméabilité de l'anastomose chez six patients sur neuf, dans un cas après 8 mois. La perméabilité des voies a été constatée chez quatre des sept patients soumis à une autopsie, et ceci jusqu'à 6 mois après avoir pratiqué une anastomose porto-cave intrahépatique par voie transjugulaire.

Surgical portosystemic venous shunting is widely used in the management of esophageal and gastric variceal bleeding secondary to portal hypertension. Regardless of the technique, the primary objective of such shunts and the lowering of intravariceal pressure is the elimination of subsequent variceal bleeding.[1,2] In the acute stage, conventional management may include vasopressin infusion, balloon tamponade and often endoscopic sclerotherapy. Emergency angiography is used by some diagnostically and therapeutically to assess patency of the portal venous system and its tributaries, and to embolize esophageal or fundal varices.[3]

The transjugular technique, now used widely for hepatic venography and manometry, and for cholangiography, liver biopsy and variceal embolization, was first described in 1967 by Hanafee and Weiner.[4,5] This route may also be applied to the nonsurgical creation of an intrahepatic portosystemic venous shunt, previously described, using the Grüntzig balloon dilatation catheter,[6,7] which can remain angiographically patent and functional. This paper reviews our experience over 4 years with transjugular intrahepatic portosystemic shunts, with particular attention to the efficacy and specific complications of this nonsurgical procedure.

Technique

Nineteen of 20 intrahepatic shunts were created as an extension of emergency angiography undertaken to confirm the presence of, and to embolize, esophageal and fundal varices, previously diagnosed by endoscopy. Abdominal aortography and selective celiac and superior mesenteric angiograms were obtained, supplemented as needed with subselective hepatic or splenic angiograms. In addition, preshunt arteriography was deemed necessary to exclude other foci of active bleeding, such as peptic ulcers, to define the anatomy and patency of the portal system, and to exclude noncirrhotic causes of portal hypertension such as hepatoma.

The procedure is depicted in Fig. 1. Once arteriography is completed, the right internal jugular vein is catheterized and a guide wire passed into the superior vena cava. A no. 9 French end-hole, curved Teflon catheter is placed through the guide wire and manipulated to the inferior vena cava and into a hepatic vein, usually in the right lobe. Wedged and free hepatic vein pressures measured manometrically evaluate corrected sinusoidal pressure. Lateral imaging is desirable to assess the anteroposterior relation of the selected hepatic vein to the portal venous system. A modified Ross needle (Cook Inc., Bloomington, Ind.) is advanced coaxially into the liver substance, 6 to 8 cm beyond the catheter tip; on slow needle withdrawal, suction with aspiration of venous blood indicates entry into the portal system and is confirmed by contrast injection. The catheter is then advanced over the needle into the portal system, usually the left portal vein, and the needle is removed.

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A curved no. 5 French catheter is subsequently placed through a guide wire into the portal venous system for manometry, angiography (Fig. 2) and embolization of varices, which is accomplished with ethanol, ethanolamine, 50% glucose, Gelfoam or Gianturco embolization coils (Cook Inc.). Following guide-wire replacement, this catheter is exchanged for a balloon dilatation catheter, which is inflated to 8 to 10 mm diameter within the tract from hepatic to portal venous system (Fig. 3). For the initial six patients previously reported[6,7] the balloon was inflated for up to 12 hours empirically, but we have since obtained similar and satisfactory results with 5 to 10 minute inflations. The no. 5 French catheter is then replaced after removing the balloon dilatation catheter, and 24 to 72 hours later follow-up manometry, angiography to assess shunt patency and further embolization as needed are carried out.

The procedure is long, often taking 4 to 6 hours and necessitating continuous monitoring of the patient; by performing the arteriography the day before shunt creation, the procedure can be shortened by 1 to 1½ hours.

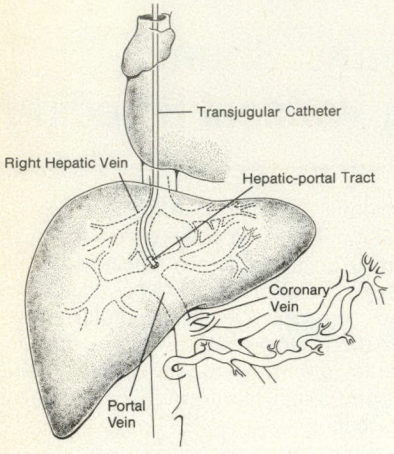

Fig. 1a

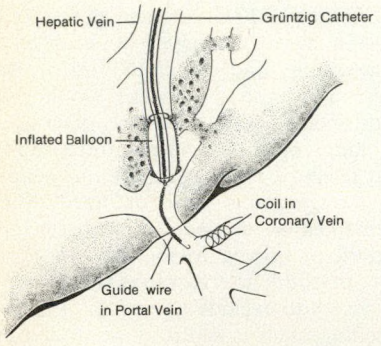

Fig. 1b

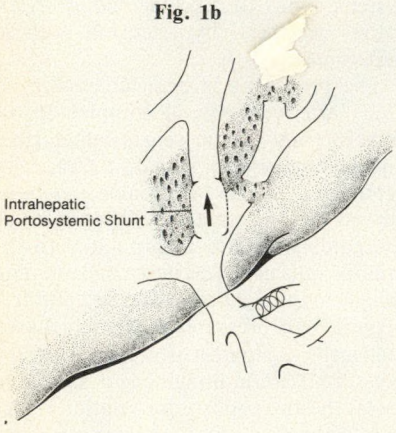

Fig. 1c

FIG. 1—Technique of transjugular intrahepatic portosystemic shunting. (a) Transjugular catheter has been advanced over modified Ross needle and tip positioned in left portal vein. (b) Balloon catheter is inflated in hepatic-portal tract. Broken lines represent split or compressed hepatic parenchyma. Coronary vein has been embolized. (c) Completed portosystemic tract with venous shunting to hepatic vein.

Patients

The records of 20 patients on whom the procedure was attempted at the Toronto General Hospital between January 1981 and April 1985 were reviewed retrospectively. There were 14 men and 6 women ranging in age from 28 to 74 years (mean 55 years). All had endoscopically proven esophageal varices and histologic confirmation of their diagnosis (alcoholic cirrhosis in 14, cryptogenic cirrhosis 3, post-necrotic cirrhosis 1, hepatitis B-related cirrhosis 1 and liver metastases from colonic carcinoma 1). The patients were placed in categories according to Child's classification[8] of liver disease (Table I).

This procedure was chosen in preference to more conventional treatment because in the majority of patients surgery was thought to be prohibitive because of end-stage liver disease. In one patient in whom two previous operative shunts had failed, this technique was selected because of anticipated surgical difficulty.

In 13 of the 20 patients the procedure was carried out as an emergency while patients were actively bleeding. Of the remaining seven, six patients were treated on an urgent basis (during hospitalization but after active hemorrhage had ceased) and one patient underwent the procedure electively. Four illustrative case reports follow.

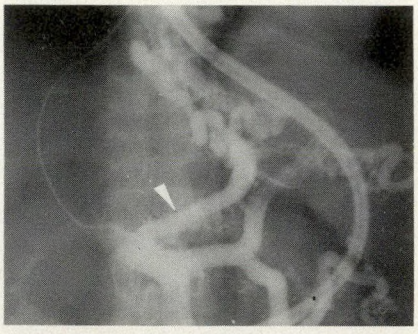

FIG. 2—Direct portogram by transjugular route demonstrates filling of portal vein tributaries and prominent coronary vein (arrow) supplying fundal and esophageal varices. Sengstaken-Blakemore tube is present.

Case Reports

Case 1

A 30-year-old man with a long history of alcohol abuse presented at another hospital with an upper gastrointestinal hemorrhage. He required a transfusion of six units of blood. A laparotomy was performed for what was presumed to be peptic ulcer disease, but a gastrotomy revealed bleeding varices. Postoperatively, he continued to bleed and was transferred to the Toronto General Hospital. Physical examination revealed marked ascites and encephalopathy. On admission, his

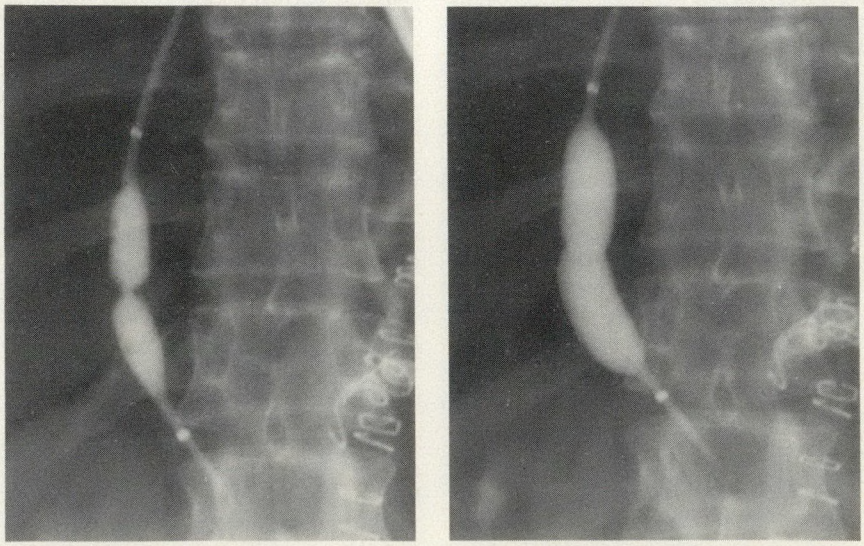

FIG. 3—Inflated balloon of Grüntzig catheter within tract from hepatic to portal veins. Initial pronounced waist-like compression of balloon represents wall of portal vein.

hemoglobin level was 95 g/L (normal 140 to 180 g/L), leukocyte count 5.2 × 10^9/L (normal 4 to 11 × 10^9/L), platelet count 90 × 10^9/L (normal 150 to 350 × 10^9/L), prothrombin time 14/11 and partial thromboplastin time 38/30. The serum aspartate aminotransferase (SGOT) level was 69 U/L (normal less than 60 U/L), serum alkaline phosphatase 64 U/L (normal less than 90 U/L), bilirubin 29 μmol/L (normal 2 to 20 μmol/L) and albumin 25 g/L (control 35 to 50 g/L). He had had several minor episodes of upper gastrointestinal bleeding and he also had a history of jaundice, mild encephalopathy and controlled ascites. There was a history of recent alcohol abuse. This man was not considered a candidate for surgery in view of the advanced stage of his liver disease.

Five days after admission he was still bleeding despite the intravenous administration of Pitressin and balloon tamponade. Coronary vein embolization was carried out and concomitantly intrahepatic portosystemic shunting.

Table I—Child's Classification of Liver Disease in 20 Patients With Attempted Transjugular Intrahepatic Portosystemic Shunting

Cause	Child's classification		
	A	B	C
Alcoholic cirrhosis	0	0	14
Nonalcoholic cirrhosis	0	1	4
Metastatic (liver) cancer	1	0	0

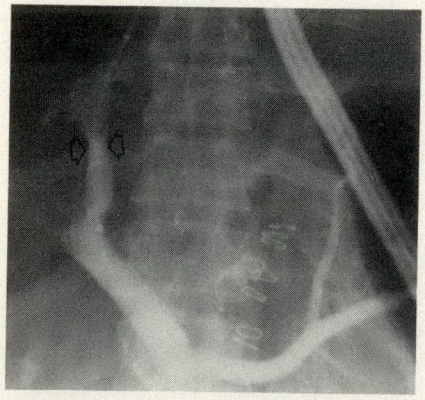

FIG. 4—Case 1. Direct portal angiogram demonstrates functioning portosystemic shunt (arrowhead) with flow of contrast cephalad into hepatic vein. Wire coil is present in coronary vein and filling of short gastric vein is seen.

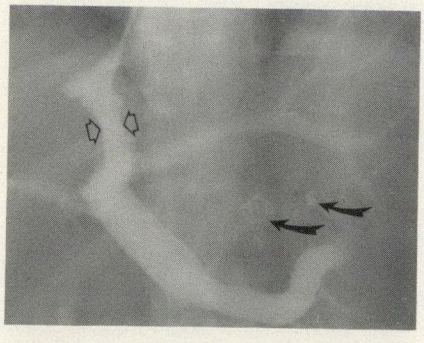

FIG. 5—Case 1. Portogram 8 months after shunt procedure demonstrates prograde portal vein flow with filling of intrahepatic portosystemic shunt (arrowheads). Curved arrows show embolization coils in coronary and short gastric veins.

Before the shunt was formed his direct portal vein pressures were 30 to 40 mm Hg. Three days later, angiography showed good filling of a patent shunt (Fig. 4). At that time, embolization of short gastric veins was performed, and portal vein pressures were 34 to 36 mm Hg. Seven days after the initial intervention, angiography revealed a patent shunt with no filling of varices and a portal vein pressure of 43 mm Hg.

Two weeks after the percutaneous shunting, he suffered minor upper gastrointestinal bleeding which was treated conservatively. He then underwent three sessions of esophageal sclerotherapy. Erosive changes were seen in the esophagus at endoscopy, and although esophageal and gastric varices were visualized, they were not thought to be the cause of bleeding. He was discharged from hospital 1 month after admission and reassessed 6 weeks later as an out-patient. The intrahepatic shunt was readily crossed with a catheter placed via an antecubital vein, and portal vein pressures were measured at 30 to 32 mm Hg. Angiography demonstrated good portal–hepatic shunting through the tract, which measured 9 mm at its minimal diameter (2 mm larger than at the time of its formation).

He has abstained from alcohol since the procedure and experienced no further gastrointestinal bleeding. At follow-up endoscopy, 8 months after the procedure, no esophageal varices were demonstrated. Portal venography and manometry again carried out through an antecubital vein, demonstrated stabilization of portal venous pressure at 31 mm Hg, no variceal filling and patency of the intrahepatic shunt, which measured 10 mm at its minimal diameter (Fig. 5). A gradient of 17 mm Hg was present across the shunt, from the portal vein (31 mm Hg) to the inferior vena cava (14 mm Hg). It is now 26 months since the shunt was performed; he has had no further bleeding, his ascites is under control and there is no evidence of encephalopathy.

Case 2

A 28-year-old man with cryptogenic cirrhosis was seen at another hospital with endoscopically proven variceal bleeding requiring five units of blood. He was treated conservatively but had persistent melena that required 16 units of blood. When transferred to the Toronto General Hospital, his hemoglobin value was 91 g/L, leukocyte count 18.1 × 10^9/L, platelet count 90 × 10^9/L, prothrombin time 11/11, partial thromboplastin time 34/30, serum aspartate aminotransferase 139 U/L, serum alkaline phosphatase 869 U/L, serum bilirubin 9 μmol/L and serum albumin 25 g/L. This man had undergone interposition mesocaval shunting in 1977 and proximal splenorenal shunting in 1978; both shunts had thrombosed, as demonstrated angiographically. His second shunt had proven technically difficult with substantial intraoperative blood loss. Because of the anticipated technical difficulties and the limited surgical options, the alternative of a transhepatic shunt was recommended and performed electively.

Ten days after transfer, he underwent transhepatic embolization of the coronary vein and the creation of an intrahepatic shunt. Before creation of the shunt, the wedged hepatic vein pressure ranged from 25 to 29 mm Hg and the free hepatic vein pressure from 11 to 15 mm Hg, resulting in a corrected sinusoidal pressure of 14 mm Hg. His direct portal vein pressure was 25 mm Hg.

Two days after the procedure, angiography demonstrated probe-patency in that the tract from hepatic to portal vein was readily crossed using only a catheter, but contrast injection into the portal system failed to demonstrate shunting to the hepatic vein. The portal vein pressure was unchanged from that measured at the time of shunt formation.

He did not bleed again and was discharged from hospital 45 days after admission. Twelve months after formation of the shunt there was no evidence of further upper gastrointestinal variceal hemorrhage, no ascites and no evidence of encephalopathy.

Case 3

A 50-year-old man with a long history and a recent episode of alcohol abuse, and with several previous episodes of upper gastrointestinal bleeding due to varices, was admitted to the Toronto General Hospital with a severe variceal hemorrhage. His hemoglobin value after resuscitation was 118 g/L, leukocyte count 26.4 × 10^9/L, platelet count 64 × 10^9/L, prothrombin time 20/10, partial thromboplastin time 110/30, serum aspartate aminotransferase 157 U/L, serum alkaline phosphatase 64 U/L, serum bilirubin 20 μmol/L, serum albumin 27 g/L. On examination, jaundice, mild encephalopathy and uncontrolled ascites were noted. Management in the past had been conservative because of his continued alcohol abuse and end-stage liver disease.

He was initially treated with Pitressin intravenously and balloon tamponade, followed by three sessions of esophageal sclerotherapy. These did not control the bleeding and after transfusion of an additional eight units of blood he underwent emergency transhepatic embolization of the coronary vein and creation of an intrahepatic shunt.

His wedged hepatic vein pressure ranged from 40 to 65 mm Hg, inferior vena caval pressure distal to the liver was 40 mm Hg, corrected sinusoidal pressure ranged from 20 to 25 mm Hg and direct portal vein pressure from 42 to 60 mm Hg. After the procedure, the upper gastrointestinal hemorrhage ceased, but the patient remained hypotensive, oliguric and comatose. Angiography 3 days after formation of the shunt revealed a drop in portal vein pressures to 37 to 55 mg Hg; his clinical condition did not improve and he died the same day. There was no evidence of further gastrointestinal bleeding after creation of the shunt.

Case 4

A 62-year-old woman with a long history of alcoholism was admitted to the Toronto General Hospital with a massive variceal hemorrhage. Her hemoglobin value was 60 g/L, serum albumin 18 g/L and serum bilirubin 13 μmol/L. Initially she was managed conservatively but the bleeding began again 5

days after admission. Not being a candidate for surgery because of her end-stage liver disease, she underwent percutaneous transjugular obliteration of the coronary vein and formation of an intrahepatic shunt. Angiography 24 hours after deflation of the balloon revealed a patent shunt with unchanged portal pressures, but with a thrombus in the portal vein. She did not bleed again and was discharged 24 days after admission with well-controlled ascites. She had a transient episode of encephalopathy which was easily reversed.

This woman died 6 months after shunt creation of a massive upper gastrointestinal hemorrhage following a prolonged bout of drinking. Autopsy demonstrated evidence of recent bleeding from esophageal varices. No gross or histologic evidence of the intrahepatic shunt was detected.

Results

Creation of the shunt was attempted in 20 patients and was successful in 15 (75%). Three of these patients rebled and after reassessment they underwent surgery. One man rebled 10 days after the procedure, even though angiography demonstrated a patent shunt, and therefore he underwent a mesocaval shunt. He recovered but died 6 months later of liver failure. A second patient rebled at 5 days; angiography revealed patency of the shunt but filling of collaterals also, and he subsequently underwent a Sugiura procedure. He bled again 5 days postoperatively and died. Autopsy revealed a patent tract from portal to hepatic vein. The third patient continued bleeding after the procedure and 24 hours later underwent emergency portocaval shunting. He continued bleeding and died the next day, despite a patent shunt.

Of the remaining 12 patients in whom the embolization and creation of the intrahepatic shunt was successful, 9 died within 30 days of the procedure (range from 0 to 20 days, mean 5.1 days). Five of the deaths were directly attributed to variceal bleeding. Three other patients died of nonvariceal bleeding (one each from erosive esophagitis, gastric ulcer and erosive gastritis). One patient stopped bleeding after the procedure but remained hypotensive with hepatorenal failure and died 3 days later.

Three patients survived more than 30 days after the procedure with no subsequent intervention. One was discharged from hospital with no evidence of rebleeding but died 6 months later of hemorrhage after excessive ethanol intake. Two other patients are alive and well 12 and 15 months after the procedure with well-controlled ascites, no encephalopathy and with no evidence of rebleeding (cases 1 and 2).

In five patients we could not create a shunt. We could not cannulate the hepatic vein in one and the portal vein branch in two others. The portal vein was thrombosed in a fourth patient, rendering the procedure impossible. In the fifth patient, the guide wire could not be advanced through the portal vein. Four of the five died of exsanguination. In the fifth patient the bleeding stopped spontaneously and the patient was discharged from hospital.

The major veins supplying the varices (usually coronary and short gastric veins) were obliterated in all patients in whom the portal system could be catheterized. Two patients had episodes of transient encephalopathy after creation of the shunt. Portal vein pressures were determined in all patients with a successfully established shunt, before the Grüntzig catheter was inflated. After shunt creation, portal vein pressures were recorded in 11 of the 15 patients, at periods ranging from a few hours up to 8 months (case 1). Preshunt pressure determinations in 15 patients ranged from 25 to 60 mm Hg (mean 40.6 mm Hg). In 11 patients, postshunt pressures decreased an average of 5.9 mm Hg (range of change from + 5 to − 14 mm Hg) (Fig. 6). In two of them it was surprising to note an increase in portal pressure of 1 mm Hg and 5 mm Hg.

In nine patients, sequential angiograms were obtained, from 12 hours to 8 months after shunt creation. One patient demonstrated thrombosis of the portal vein and presumably the shunt also. In two patients probe-patency but no radiologically demonstrable flow was noted. In the remaining six flow was visualized through a patent shunt. Follow-up portal venograms could not be obtained in six patients.

Autopsy was performed on seven patients. No gross or histologic evidence of the intrahepatic shunt could be demonstrated in three, but on gross examination the remaining four had patent tracts between the portal and hepatic veins. Pseudointima lining the tract was visualized histologically in two of these patients, at 3 weeks and 6 months after creation of the shunt (Fig. 7).

Complications

Intraperitoneal bleeding occurred in one patient who died less than 24 hours after the procedure of gastrointestinal bleeding, which antedated shunting and persisted thereafter. At autopsy, he was found to have erosive gastritis with blood in the bowel; 8.5 L of admixed blood with ascitic fluid was found in the peritoneal cavity. Capsular perforation of the liver was presumed but not confirmed. Sepsis

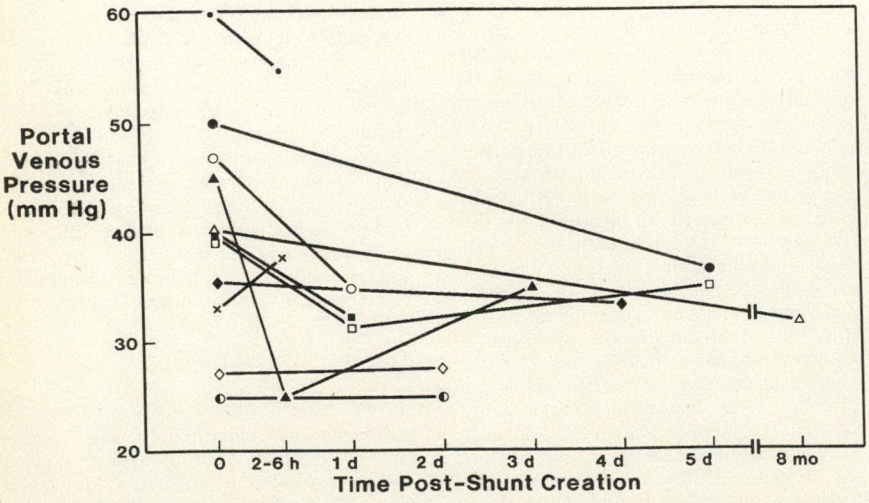

FIG. 6—Time versus pressure curves of 11 patients with sequential direct portal vein pressures.

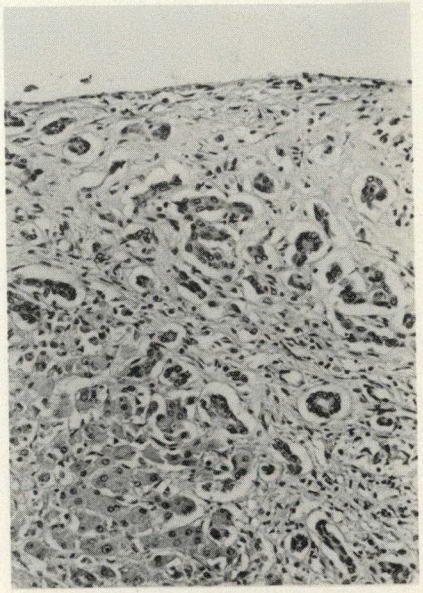

FIG. 7—Wall of intrahepatic shunt demonstrates neointima covered by flattened cells, continuous with underlying cirrhotic liver (hematoxylin and eosin, original magnification × 250).

was noted in two patients early in the series, but they had other indwelling lines as well. Thrombosis in part or all of the portal vein was observed in two patients, again early in the series when the balloon catheter was left inflated in the tract for longer periods, with the tip protruding into the portal system.

Two of the surviving patients had transient episodes of encephalopathy.

Discussion

Treatment of portal hypertension with bleeding varices continues to rely predominantly on the operative creation of portosystemic shunts.[3,9–11] Optimally, such procedures should be performed electively, due to the undesirable high operative death rate (40% to 80%) reported for emergency procedures.[2,12,13]

Traditional operative approaches include central, nonselective (e.g., end-to-side portocaval), peripheral nonselective (e.g., mesocaval and standard splenorenal) and peripheral selective (e.g., Warren) shunts. All have the common aim of primary decompression of all or part of the portal venous system. No one approach is clearly superior when assessed by long-term survival.[2,10–14] Although differences in postoperative morbidity do exist, selective shunts such as the Warren distal splenorenal shunt appear to be associated with less postoperative encephalopathy, probably because prograde portal flow is preserved.[10,15] This quality is also theoretically desirable for the hepatotrophic factors that portal blood carries, which may aid liver regeneration and maintain hepatocyte integrity.[10,15,16]

The successful intrahepatic shunt is unique since it allows a complete shunt (i.e., diversion of portal flow nonselectively) while, at least theoretically, promoting prograde portal flow; this property has been demonstrated by others[17] to be associated with a substantially lower incidence of portosystemic encephalopathy than occurs in patients with reversed flow (11% versus 55% respectively). In the present series, 2 of 15 patients with a successfully created shunt demonstrated mild transient encephalopathy. Patients who undergo shunt procedures that totally divert portal flow away from the liver are most likely to suffer encephalopathy.[1] To date, we have used empirically a 9 to 10-mm diameter balloon. A larger tract might be expected to lower pressure further, but this might result in excessive diversion of portal flow which would promote encephalopathy and at the same time reduce hepatic perfusion, thus furthering hepatic failure.

Sarfeh and colleagues[18] created portocaval shunts as small as 10 mm diameter in conjunction with collateral ligation and achieved prevention or arrest of variceal bleeding in spite of angiographically visualized persistent collaterals. However, they expressed reservations regarding the long-term patency of these small shunts. Angiographically, we have observed that in patients with more favourable Child's classifications (and presumably more "normal" livers) the shunt is more easily created (i.e., the balloon inflates with much less pressure), but that the recorded shunt size is smaller than the 10 mm diameter of the inflated balloon. Perhaps the natural resiliency of a less cirrhotic liver is more likely to close the tract, or to leave it probe-patent as we observed in two patients with Child's classes A and B disease. The tracts in these patients, at follow-up angiography, were readily crossed with only a catheter, but no portal–hepatic shunting could be demonstrated and repeated balloon dilatation produced no further increase in the diameter of the tract. However, we have not yet devised a technique for placing a suitably-sized stent or strut to maintain tract patency.

Thrombosis in the portal vein or its intrahepatic branches was observed in two patients early in the series when the balloon catheter was left inflated in the tract, with the catheter tip in the portal vein, for up to 12 hours. This tip may have functioned as a thrombogenic nidus, as we have not observed portal vein thrombosis since limiting dilatation time to only 5 to 10 minutes.

In this series, the calculated average decrease in portal vein pressure of 5.9 mm Hg may be an underestimate for two reasons. First, the pressure in many patients was initially obtained while they were actively bleeding and hypovolemic, and may not accurately reflect pressures in the normovolemic state. Second, and more important, we have hitherto determined the baseline portal vein pressure before variceal embolization, which can be expected to raise portal venous pressures. This is supported by the data of others[18] who have recorded increases in gradients across shunts after surgical occlusion of collaterals by ligation.

Most patients in our series were referred to the radiologist for angiographic obliteration of varices; the intrahepatic shunt was created as an extension of this therapy. It was performed initially only as an emergency procedure in patients with end-stage cirrhosis and a poor prognosis according to Child's classification and in those with life-threatening gastrointestinal bleeding, who were not candidates for emergency shunt surgery. Later in the series, however, as we became familiar with the technique, we began to use it as an alternative in the management of portal hypertension and, in fact, it was used electively in one of our patients. It served as a life-saving, temporizing maneuver in three patients who later underwent surgical intervention. Of three other patients discharged from hospital after the procedure, one had adjunctive sclerotherapy.

Although the overall results in this small series are not impressive, it must be recognized that for most patients, the formation of an intrahepatic shunt in conjunction with embolization of the coronary vein and varices was a final life-saving procedure after all others had failed. In better-risk patients, one would expect better survival rates. Moreover, technical improvements are still required both to decrease the failure rate of the procedure and to maintain long-term shunt patency and flow. It must be emphasized that this procedure is not meant to replace any of the existing and accepted treatments for bleeding esophageal varices; instead we suggest that it may prove to be a useful adjunct.

References

1. WEESE JL, YALE CE, PELLETT JR, et al: Shunts for portal hypertension. *Am Surg* 1983; 49: 365–368
2. STEEGMÜLLER KW, MÄRKLIN HM: The portocaval shunt in the treatment of portal hypertension. *Br J Clin Pract* 1984; 38: 171–175
3. HANNA SS, WARREN WD, GALAMBOS JT, et al: Bleeding varices: 1. Emergency management. *Can Med Assoc J* 1981; 124: 29–41
4. HANAFEE W, WEINER M: Transjugular percutaneous cholangiography. *Radiology* 1967; 88: 35–39
5. WEINER M, HANAFEE W: Review of transjugular venography. *Radiol Clin North Am* 1970; 8: 53–68
6. COLAPINTO RF, STRONELL RD, BIRCH SJ, et al: Creation of an intrahepatic portosystemic shunt with a Grüntzig balloon catheter. *Can Med Assoc J* 1982; 126: 267–268
7. COLAPINTO RF, STRONELL RD, GILDINER M, et al: Formation of intrahepatic portosystemic shunts using a balloon dilatation catheter: preliminary clinical experience. *AJR* 1983; 140: 709–714
8. CHILD CG III: *Hepatic Circulation and Portal Hypertension*, Saunders, Philadelphia, 1954
9. ARCHIE JP JR, FELDTMAN RW: Semi-elective portal systemic shunts for variceal bleeding. *South Med J* 1981; 74: 1211–1212
10. GUSBERG RJ: Shunts for variceal hemorrhage: why? when? what? *Surg Clin North Am* 1980; 60: 1265–1272
11. BUSUTTIL RW: Selective and nonselective shunts for variceal bleeding. A prospective study of 103 patients. *Am J Surg* 1984; 148: 27–35
12. REINER DS, KAMINSKI DL: Comparative evalution of selective and nonselective peripheral portosystemic shunts for treatment of variceal hemorrhage. *Am J Surg* 1982; 144: 704–710
13. CELLO JP, DEVENEY KE, TRUNKEY DD, et al: Factors influencing survival after therapeutic shunts. Results of a discriminant function and linear logistic regressions analysis. *Am J Surg* 1981; 141: 257–265
14. REZNICK RK, LANGER B, TAYLOR BR, et al: Results and hemodynamic changes after interposition mesocaval shunt. *Surgery* 1984; 95: 275–280
15. OROZCO H, GUEVARA L, URIBE M, et al: Survival and quality of life after selective portasystemic shunts. *Am J Surg* 1981; 141: 183–186
16. RIKKERS LF, MILLER FJ, CHRISTIAN P: Effect of portasystemic shunt operations on hepatic portal perfusion. *Ibid:* 169–174
17. RYPINS EB, SARFEH IJ: Does portal pressure influence direction of portal flow and encephalopathy rates after 10-mm portacaval shunts in man? *J Surg Res* 1984; 37: 119–122
18. SARFEH IJ, RYPINS EB, CONROY RM, et al: Portacaval H-graft: relationships of shunt diameter, portal flow patterns and encephalopathy. *Ann Surg* 1983; 197: 422–426

CAMILLE M. EL KHATIB, FRCS

Strangulated Femoral Hernia Containing Acute Gangrenous Appendicitis: Case Report and Review of the Literature

The author reports the case of a 69-year-old woman with acute gangrenous appendicitis in a stangulated right femoral hernia. The hernia was diagnosed preoperatively, but the gangrenous appendix was found in a strangulated loop of small bowel only at operation. The combination of gangrenous appendicitis and strangulated loop of small bowel in a femoral hernial sac is an unusual finding, but awareness of it will avoid delay in treatment and lead to the patient's full recovery.

L'auteur décrit le cas d'une femme de 69 ans souffrant d'une appendicite gangréneuse aiguë dans le contexte d'une hernie fémorale droite étranglée. La hernie avait été diagnostiquée en préopératoire, mais on ne découvrit l'appendice gangréneux dans l'étranglement d'une anse de l'intestin grêle qu'au moment de l'opération. Il est rare qu'on observe l'association d'une appendicite gangréneuse et d'un étranglement de l'intestin grêle dans un sac herniaire fémoral; sa recherche permettra d'éviter les délais de traitement et d'obtenir le rétablissement éventuel complet du malade.

Incarcerated appendix in a femoral hernia is an unusual finding and unlikely to be diagnosed preoperatively. A case is reported below in which the patient had a strangulated hernia containing a gangrenous appendix.

Case Report

A 69-year-old woman complained of abdominal pain, nausea, vomiting and abdominal distension for 3 days.

Her body temperature was 39°C. The abdomen was distended and tender with increased bowel sounds. There was a swelling in the right groin that was tender without impulse on coughing. The overlying skin was red and edematous, with extension into the right inguinal region and labium majus.

Her hemoglobin level was 150 g/L, the leukocyte count 14.3×10^9/L with a neutrophil leukocytosis, and the serum potassium concentration was 2.9 mmol/L. An abdominal x-ray film showed dilated loops of small bowel with fluid levels suggesting a subacute obstruction.

A diagnosis of inflamed strangulated femoral hernia was made. The operative approach was through a vertical incision made over the femoral canal and continued upwards above the inguinal ligament (McEvedy approach). A gangrenous appendix and dark red loop of small bowel were found together, with foul-smelling yellow fluid in the inflamed hernial sac. The fluid was sucked out and the bowel wrapped with a warm moist abdominal pack until the colour improved. It was then reduced and the femoral ring repaired from above. The peritoneum was opened from above and routine appendectomy performed followed by noxythiolin lavage.

The woman's postoperative course was uncomplicated and she was discharged a week later.

The pathology report on the operative specimen was "acute gangrenous appendicitis". The appendix was acutely inflamed and a fecalith was present.

Discussion

The first case of incarcerated appendix in a femoral hernia was reported in 1731 by De Garengot.[1] In 1962 Kia-Nouri[2] reviewed 233 cases of femoral hernia containing incarcerated appendix, but in very few was there evidence of appendicitis. The frequency of acute appendicitis in a femoral hernia has not been reported, but Ryan[3] found 11 (0.13%) of 8692 cases of acute appendicitis in external hernias. The same percentage was found by Lester and Bourke.[4] This condition is most frequently seen in postmenopausal women.[4] Constriction of the lumen of the appendix by the neck of the hernial sac[5] occurs, leading to inflammation and gangrene of the appendix. This case illustrates a rare combination of gangrenous appendicitis and strangulated loop of small bowel.

Once the appendix becomes inflamed, the narrow neck of the hernial sac prevents spread of the infection and generalized peritonitis.[6] The presence of normal appendix within the hernia is unlikely to be diagnosed preoperatively,[7] but in an inflamed appendix the association of a tender right-sided hernia with few constitutional symptoms[8] and the presence of redness and edema overlying the skin of the groin may be suggestive.[7] Pyrexia and leukocytosis are not constant findings in hernial appendicitis,[8] although both were present in our case. Awareness of the existence of this rare and serious condition avoids delay in management and will make for an uneventful recovery.

I thank Mr. G.J.A. Brown, consultant general surgeon, for his help.

References

1. GARLAND EA: Femoral appendicitis. *J Indiana Med Assoc* 1955; 48: 1292–1294

2. KIA-NOURI M: Associated incarcerated appendix in a femoral hernia. *Journal of the Albert Einstein Medical Center* 1962; 10: 38–39

3. RYAN WJ: Hernia of the vermiform appendix. *Ann Surg* 1937; 106: 135–139

4. LESTER R, BOURKE JB: Strangulated femoral hernia containing appendices. *J R Coll Surg Edinb* 1979; 24: 102–103

5. JOHNSON CD: Appendicitis in external herniae. *Ann R Coll Surg Engl* 1982; 64: 283

6. WATKINS RM: Appendix abscess in a femoral hernial sac — case report and review of the literature. *Postgrad Med J* 1981; 57: 306–307

7. SPENCER RF, LIVINGSTONE PD: Unusual combined pathology in a femoral hernia. *J R Coll Surg Edinb* 1984; 29: 255–256

8. THOMAS WE, VOWLES KD, WILLIAMSON RC: Appendicitis in external herniae. *Ann R Coll Surg Engl* 1982; 64: 121–122

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A Biomechanical Study of Wire Fixation

A biomechanical study of wire fixation was performed using 18-gauge stainless steel wire on an Instron universal testing machine. Six groups of wires were tested — an intact piece of wire (control), a wire bent to a right angle five times, a simple knot, a square knot, a loop knot and a twist knot. The tensile load-to-failure value for each wire was recorded. The intact wire broke at a mean load of 67 kg. The wires with the bend, the simple knot and the square knot all broke at similar loads. Those wires with a knot broke at the knot. The loop and the twist knots pulled apart at much lower loads. These results indicate that bending the wire does not weaken it substantially, but kinking may initiate the site of breakage. The square knot is the strongest knot; the loop and twist knots are not recommended if the wire is to be under tension.

Les auteurs ont réalisé une étude biomécanique de la fixation par fil métallique — un fil d'acier inoxydable a été testé sur une machine universelle Instron. On a étudié cinq échantillons de six différentes sortes de fil — un fil intact, un fil plié cinq fois à angle droit, un fil avec un noeud simple, un avec un noeud plat, un avec un demi-noeud gansé et enfin un avec destours morts. Ils ont mesuré à quelle charge les fils cassaient ou les noeuds se défaisaient. Les fils intacts ont cassé pour une traction moyenne de 67 kg. Les fils avec les pliures, les noeuds simples et les noeuds plats se sont rompus pour des charges similaires. Ceux avec un noeud se sont cassés au niteou du noeud. Les demi-noeuds gansés et à tours morts se sont défaits pour des charges beaucoup plus faibles. Ces résultats montrent que plier le fil ne l'affaiblit pas notablement mais qu'une torsion peut être le régede la cassure. Le noeud plat est le plus résistant et les demi-noeuds cinq gansés et à tours morts ne olevraient probablement pas être utilisés si le fil doit être sous tension.

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The use of wire for internal fixation is common in orthopedic practice. It may be used as cerclage or as a tension band in a variety of situations such as in oblique fractures of long bones, spinal fixation (Fig. 1a), osteotomized trochanters (Fig. 1b) and in patellar fractures (Fig. 1c). As with any form of inter-

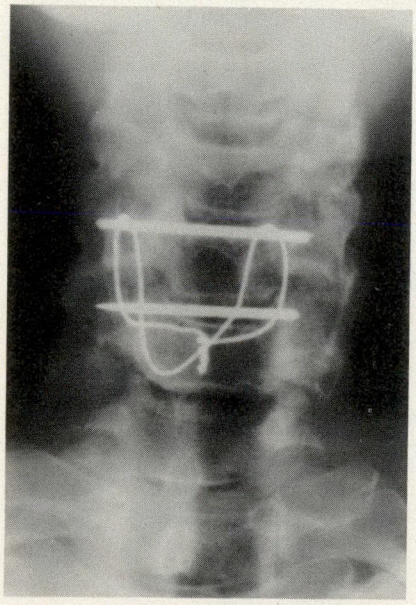

Fig. 1a

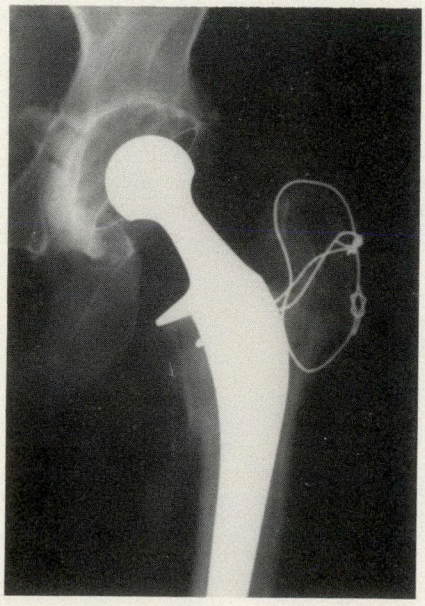

Fig. 1b

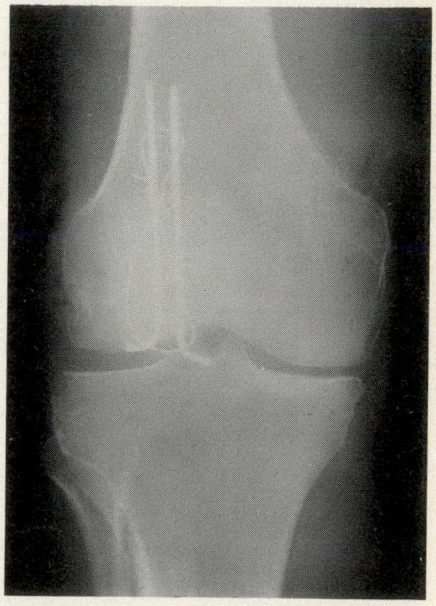

Fig. 1c

FIG. 1—Surgical uses of wire fixation: (a) spinal fixation; (b) reattachment of osteotomized trochanter; (c) patellar fracture fixation.

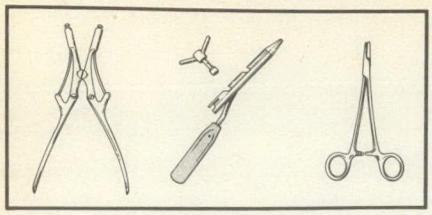

FIG. 2—Instruments used for tying knots in wire (left to right): Harris knotter, Synthes knotter, needle driver.

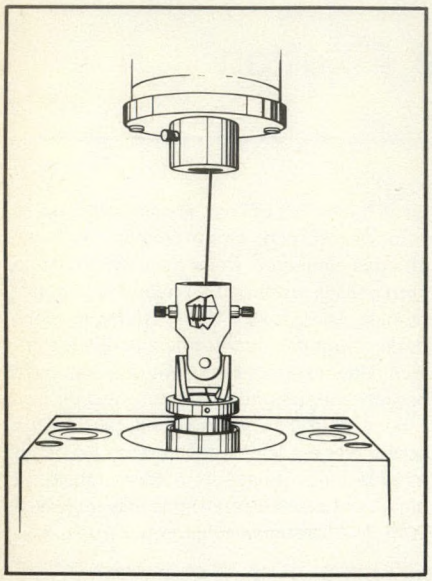

FIG. 3—Lengths (15 cm) of 18-gauge wire were mounted in Instron 1125 universal testing machine and strained at rate of 5 mm/s.

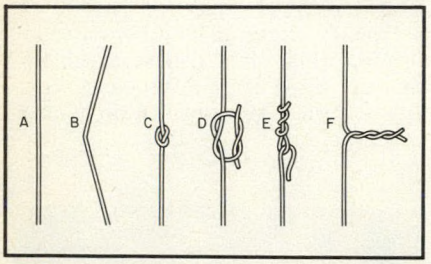

FIG. 4—Wires and knots tested: (A) intact length of wire (control); (B) wire bent to right angle five times before testing; (C) intact wire with simple knot; (D) wire with square knot, tied by Harris knotter; (E) wire with loop knot; (F) wire with twist knot — three complete twists made by needle driver.

nal fixation, the wire may break before healing has been achieved, from fatigue, the characteristics of the metal alloy or the manner in which the wire was fastened.

Various methods are used to secure wires (Fig. 2). The purpose of this study was to determine the best method of tying knots in the wire and what effect handling of the wire had on its ultimate strength.

Materials and Method

Stainless steel wire (18-gauge) was tested on an Instron 1125 universal testing machine (Instron Corp., Canton, Mass.). A 150-mm length of wire was fastened over two steel rods and with the crosshead moving at a rate of 5 mm/s, a tensile load was applied (Fig. 3).

Six different groups of wire were tested (Fig. 4).

- An intact length of wire (control) (Fig. 4A).
- A piece of wire that was bent in the middle to a right angle five times before testing (Fig. 4B).
- An intact piece of wire with a simple knot in the middle (Fig. 4C).
- A wire with a square knot in the middle, tied by a Harris knotter (Fig. 4D).
- A piece of wire with a loop-knot in the middle (Fig. 4E).
- A twist knot in the middle of the wire using a needle driver to make three complete twists (Fig. 4F).

The force at which the wires failed, or at which the knots pulled apart, was recorded and the mean load-to-failure value was calculated. Five wires were tested in each of the six groups.

Results

The intact control wire broke at a mean tensile load of 67 kg. Compared with the control, bending the wire before testing did not weaken it and the site of failure was not at the bend. The wire with the simple knot was a strong as the virgin wire but broke at the knot in four of the five wires tested. The wire with the square knot withstood a mean load of 66 kg but again failed at the knot in all five cases. The wire with the loop knot did not break but pulled apart at a mean load of only 25 kg. The wire with the twist knot had completely unravelled at a mean load of 22 kg (Fig. 5).

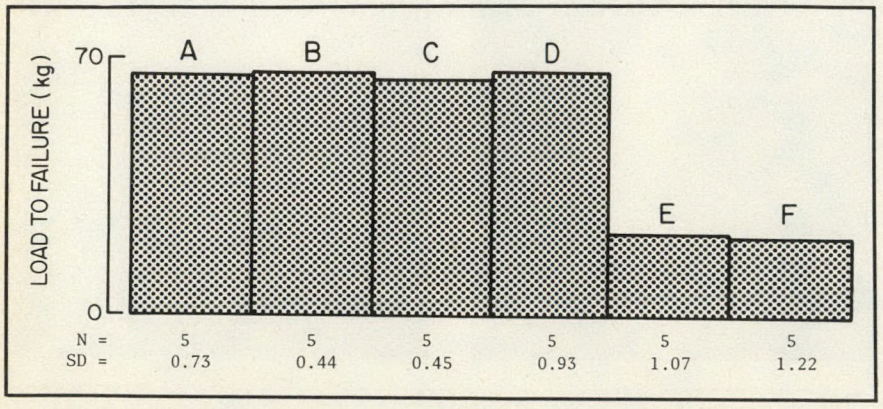

FIG. 5—Load-to-failure values for each test group as in Fig. 4. Due to highly repeatable results, standard deviations were small and are reported below each group.

Discussion

The results indicate that bending the wire during application does not significantly decrease the load-to-failure value, but previous studies have shown that bending may decrease fatigue strength.[1] Metallurgically, it is known that such cold-working on metal will increase its yield-load, decrease the strain-to-failure and leave the failure-load unchanged. Kinking of the wire during application, as simulated by the simple knot, may be the site of breakage and should be avoided.

The square knot was the strongest of the three knots tested whereas the loop knot and twist knot pulled apart at low tensile loads. In an earlier study, Schultz and colleagues[2] found the twist knot to be superior to both the square and loop knots, but they calculated the yield point and not the load-to-failure value. Our results were similar to those of Wang and colleagues.[3]

Conclusions

Twist knots and simple loop knots are unsuitable for load-bearing applications, as they separate at tensile loads of one-third of those achieved with the more conventional square knot. A knot does not produce a significant change in the tensile strength of the virgin wire. Similarly, repetitive flexing (kinking) of the wire, as many as five times, does not change the tensile strength; however, it is recognized that such flexing will have an effect upon the fatigue strength of the wire. Based on our data, we do not recommend the loop or twist knots if the wire is to be subjected to high tensile forces.

References

1. OH I, TREHARNE RW, SANDER T: A wire fatigue tester and the strengths of various orthopaedic wires. *Trans Orthop Res Soc* 1982; 7: 312

2. SCHULTZ RS, BOGER JW, DUNN HK: Strength of stainless steel surgical wire in various fixation modes. *Clin Orthop* 1985; 198: 304–307

3. WANG GJ, REGER SI, JENNINGS RL, et al: Variable strengths of the wire fixation. *Orthopedics* 1981; 5: 435–436

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Diplopia and Diabetes Insipidus Secondary to Type II Fracture of the Sella Turcica: Case Report

Fractures of the sella turcica are rare and are often difficult to diagnose radiologically. They can produce a wide variety of complications, including septic, neuroendocrine, neurovascular and neuro-ophthalmologic problems. The authors describe the case of a 17-year-old boy with diabetes insipidus and diplopia secondary to a type II fracture of the sella turcica. They emphasize the clinical importance of fractures in the sellar region in view of their proximity to vital structures and they discuss possible mechanisms of causation.

Les fractures de selles turciques sont rares et souvent difficiles à diagnostiquer à la radiographie. Elles peuvent produire un grand nombre de complications: problèmes infectieux, neuroendocriniens, neurovasculaires et neuro-ophtalmologiques. Les auteurs décrivent le cas d'un garçon de 17 ans frappé de diabète insipide et de diplopie des suites d'une fracture de type II de la selle turcique. Ils soulignent l'importance clinique des fractures de la région sellaire, compte tenu de leur proximité avec des structures vitales, et ils commentent les mécanismes étiologiques possibles.

Fractures of the skull base are not uncommon in blunt head trauma.[1] The majority of fractures are extensions of fractures of the temporal bone or skull base and usually elude accurate radiologic localization.[2-4] Thus, clinical signs may be the sole indication of their presence. Fractures in the region of the sella turcica are usually associated with severe blunt head injury and have a propensity for producing a broad spectrum of neuro-ophthalmologic, neuroendocrine, cerebrovascular and septic complications.[4-7] Only 29 cases of these rare fractures (traumatic and spontaneous) have been described in the literature to date.[2,4,5,8,9] They are classified into three types: type I is confined to the dorsum of the sella, type II involves the floor of the sella and type III extends through the base to the sphenoid sinus and the clivus (Fig. 1). The majority of sellar fractures reported in the literature are complex type III fractures.

This is a report of a boy who had diplopia and diabetes insipidus after blunt head injury, resulting in a type II fracture of the sella turcica and fracture of the right mandible.

Case Report

A 17-year-old boy was admitted to the University Hospital of the West Indies after falling approximately 10 m from a tree. Subsequently he experienced transient loss of consciousness and a seizure. On admission, he was bleeding from the ears and nose and was disoriented as to time and place.

X-ray films showed fractures of the right mandible and right clavicle. Skull films were reported as normal. Within 24 hours of admission diabetes insipidus developed, with urine volumes exceeding 5 L/d and a specific gravity of urine ranging from 1.000 to 1.002. Right nervus abducens palsy developed with diplopia on right lateral gaze. At the time of discharge his polyuria and polydipsia had resolved but his diplopia remained unchanged.

On follow-up 1 month later his diplopia had improved. Review of the skull x-ray films revealed a displaced fracture of the floor of the sella turcica with a fluid level in the sphenoid sinus (Fig. 2).

Discussion

Fractures of the sella turcica are rare.[2] Their exact incidence with blunt head injury is unknown. Dublin and Poirier[5] found only five cases (1.4%) in 350 consecutive skull fractures. However, their preponderance in fatal craniocerebral trauma is becoming increasingly recognized, and it is estimated that such fractures occur in about 20% of severe head injuries and are associated with a high death rate, because of extensive brain damage.[4]

These fractures occur most commonly as an extension of other fractures at the base of the skull and have a close correlation with frontal and maxillofacial trauma.[1,2,4,5] Several mechanisms have been suggested as the cause of fractures

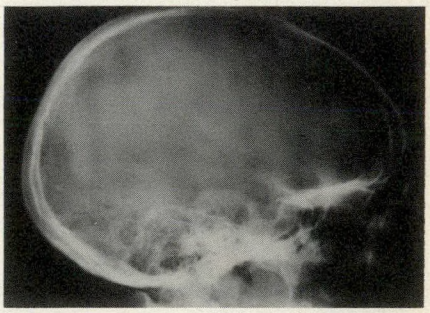

FIG. 2—Lateral skull x-ray film showing displaced fracture of floor of sella turcica (type II) with air fluid level in sphenoid sinus.

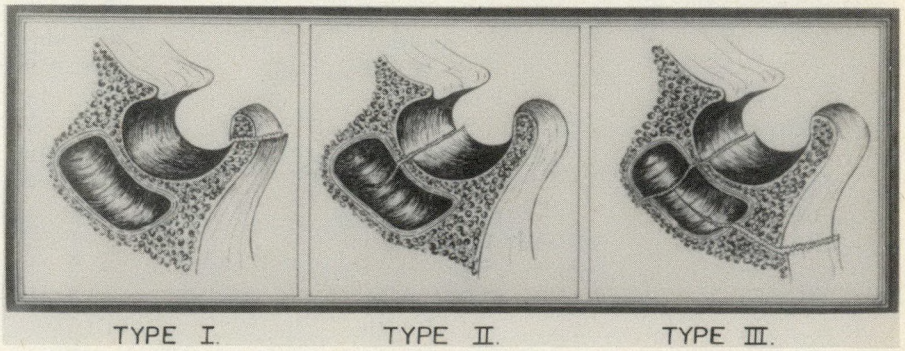

FIG. 1—Schematic diagrams demonstrating main clinical types of fractures found in sellar region in clinical practice.

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in the sellar region. One is the traumatic displacement of the middle third of the facial skeleton and traumatic dislocation of the mandible secondary to maxillofacial trauma.[4,10] In such cases sellar fracture can result from the transitory impact of the displaced facial bone against the base of the skull, and was probably the mechanism of injury in our case. Fractures of the frontal bone resulting from direct impact tend to extend inferiorly along the floor of the anterior cranial fossa into the sella. Experimental studies have also shown that fractures of the sella may accompany midline occipital fractures that extend to the foramen magnum.[1] It is apparent that any fracture of the cranial vault, extending caudally to the skull base, is capable of reaching the sellar region,[4] a fact well demonstrated in autopsy studies of fractures in the floor of the anterior and middle cranial fossae.[3] Hence, isolated fractures of the sella are rare in head injury, but isolated type I fractures and fractures of the anterior clinoid have been described.[3] Such fractures occurring in blunt head trauma are probably due to transmission of shearing forces through the petroclinoid ligament.[4]

In most cases[5,6] fractures in the sellar region are easily detected on a plain lateral roentgenogram of the skull, because by extending into the sphenoid bone they are always accompanied by opacification of the sphenoid sinus from hemorrhage.[1] Other signs include pneumocephalus or intraventricular air. Tomography is invaluable in confirming cases with equivocal features. In view of the proximity of the sella to the optic apparatus, cranial nerves III to VI, the cavernous sinus, carotid siphon basilar artery, paranasal sinuses, hypothalamus and the pituitary gland, fractures in this region are associated with a broad spectrum of complications. They include injury to cranial nerves III to VI, cerebrospinal fluid rhinorrhea, otorrhea, basilar artery entrapment,[11] spasm or occlusion with consequent cerebral ischemia, caroticocavernous fistula, pituitary disturbance and meningitis.[2,4–6] The cranial nerve palsies may be temporary or permanent. Because of its intracavernous course, unlike the other nerves that travel in the wall of the cavernous sinus, cranial nerve VI is the most frequently injured with sellar fractures and the concomitant diplopia from lateral rectus palsy in our patient was probably a result of such injury. A few cases of injury to the optic nerve, chiasm and vestibuloacoustic and facial nerves have also been documented.[4,6,7] Anterior and posterior pituitary disturbances (manifesting as diabetes insipidus) have been described in association with fractures of the sella. The diabetes may manifest itself several months after the accident.[6,12]

Conclusions

Awareness of fractures in the sellar region is important in view of its proximity to several vital structures. Early detection is essential for prompt recognition of the numerous complications (some of which can mimic intracranial mass lesions) to avoid unnecessary surgery. Patients found to have a fracture of the sella turcica should be placed under surveillance because of the possible development of septic, neuroendocrine, neurovascular or neuro-ophthalmologic complications.[5]

References

1. REYNOLDS DF: Traumatic effusion of the sphenoid sinus. *Clin Radiol (Lond)* 1961; 12: 171–176
2. CAGLAR MK, CEYHAN M, SENSES DA, et al: Radiological case of the month. Fracture of the sella turcica. *Am J Dis Child* 1984; 138: 605–606
3. SPIERS F: Isolated fracture of the dorsum sellae. *Acta Ophthalmol* 1964; 42: 884–888
4. YOUNG HA, OLIN MS, SCHMIDEK HH: Fractures of the sella turcica. *Neurosurgery* 1980; 7: 23–29
5. DUBLIN AB, POIRIER VC: Fracture of the sella turcica. *Am J Roentgenol* 1976; 127: 969–972
6. ENGELS EP: Basal skull fractures involving the sella turcica. *Clin Radiol (Lond)* 1961; 12: 177–178
7. RESNECK JD, LEDERMAN IR: Traumatic chiasmal syndrome associated with pneumocephalus and sellar fracture. *Am J Ophthalmol* 1981; 92: 233–237
8. DU BOULAY GH, EL GAMMAL T: The classification, clinical value and mechanism of sella turcica changes in raised intracranial pressure. *Brit J Radiol* 1966; 39: 422–442
9. GUTIN PH, CUSHARD WG JR, WILSON CB: Cushing's disease with pituitary apoplexy leading to hypopituitarism, empty sella, and spontaneous fracture of the dorsum sellae. Case report. *J Neurosurg* 1979; 51: 866–869
10. CARLSON GO, HAVERLING M, MOLIN C: Isolated fracture of the base of the skull within the sella region. *Acta Radiol [Diagn] (Stockh)* 1973; 14: 662–666
11. LOOP JW, WHITE LE JR, SHAW CM: Traumatic occlusion of the basilar artery within a clivus fracture. *Radiology* 1964; 83: 36–40
12. BISTRITZER T, THEODOR R, INBAR D, et al: Anterior hypopituitarism due to fracture of the sella turcica. *Am J Dis Child* 1981; 135: 966–968

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Bilateral Lower Extremity Amputations After Prolonged Application of the Pneumatic Antishock Garment: Case Report

The authors describe the case of a 29-year-old man with multiple trauma who suffered compartment syndromes necessitating bilateral lower limb amputations as a result of the prolonged (9.5 hours) application of a pneumatic antishock garment (PASG). There was no evidence of lower limb trauma before the garment was put on. Despite the apparent benefits of the PASG in traumatized hypovolemic patients, the lowest possible inflation pressures should be used and removal attempted as soon as hemodynamic stability can be assured.

Les auteurs décrivent le cas d'un homme polytraumatisé de 29 ans qui a souffert d'un syndrome des loges des extrémités inférieures nécessitant une amputation bilatérale des membres des suites de l'utilisation prolongée (9.5 heures) d'un vêtement pneumatique antichoc (VPAC). Avant l'emploi du vêtement, on n'avait constaté aucun signe de traumatisme des membres inférieurs. Malgré les avantages apparents du VPAC chez les patients traumatisés hypovolémiques, le gonflement le plus faible est de mise et il y a lieu de tenter de le retirer aussitôt qu'une stabilité hémodynamique semble assurée.

External pneumatic counterpressure has been used since 1909, when Crile[1] first described it in resuscitating a pulseless victim with a penetrating neck injury. Although the proposed mechanisms of action (tamponading of bleeding sites, autotransfusion, increased peripheral vascular resistance) remain unclear,[2-5] and there are no well-controlled clinical studies demonstrating a favourable outcome after its use,[6,7] the pneumatic antishock garment (PASG) continues to be recommended for traumatized patients in shock. Complications of the PASG include reduced renal function, lactic acidosis, respiratory compromise, exacerbation of congestive heart failure and increased intrathoracic bleeding.[8-12] A recently recognized complication with potential long-term morbidity is that of lower extremity compartment syndromes,[8,13-17] as described below.

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Case Report

A 29-year-old man was struck by a train. Initially he was taken to a local hospital where he arrived in a state of shock. Management included the administration of crystalloid solution, transfusion of type-specific blood and insertion of a right-sided chest tube for hemothorax. The leg compartments of the PASG were inflated 70 minutes after injury, and when a peritoneal lavage was grossly positive for blood the abdominal compartment was also inflated. There was no clinical or radiologic evidence of lower extremity trauma before application of the PASG; the type of suit did not allow monitoring of pressure within the compartments.

He was referred to the Regional Trauma Unit at Sunnybrook Medical Centre. On arrival his blood pressure was 80/40 mm Hg, the heart rate was 135/min and there was very little urine output (less than 10 ml/h). Fluid resuscitation continued, with crystalloid solution and typed unmatched blood. Examination and x-ray films revealed fractures of the right radius and ulna, a compound fracture of the right humerus, a large right temporal-parietal scalp laceration and a Glasgow Coma Scale value of 4. Computerized tomography revealed only a small right temporal contusion. At laparotomy, 1 hour after arrival, his systolic blood pressure was 90 mm Hg despite the administration of 14 units of packed red blood cells, 4 units of fresh frozen plasma and 4 L of crystalloid solution. A laceration of the right anterior segment of the liver with devitalized tissue was treated by resectional débridement.

In the operating room a further 10 units of packed red blood cells, 10 units of fresh frozen plasma, 12 units of platelets and 6 L of crystalloid solution were given. The systolic blood pressure throughout the intraoperative period ranged between 60 and 100 mm Hg. Furosemide, mannitol and low-dose dopamine were administered but failed to increase the urine output. Throughout the period of resuscitation and operation the leg compartments of the PASG remained inflated. An attempt at reducing the pressure in the leg compartments toward the end of the operation resulted in a fall in blood pressure to 80 mm Hg (from 100 mm Hg), so the compartments were reinflated.

When the patient was transferred to the intensive care unit 10 hours after injury, his blood pressure was 110/60 mm Hg. The leg compartments of the PASG were gradually deflated and with the blood pressure stable at 110/80 mm Hg the garment was totally removed 1 hour later; the total duration of inflation was 9.5 hours. The lower extremities appeared normal and distal pulses were present.

During the next 48 hours the patient suffered complications resulting from the prolonged hypotension. Acute tubular necrosis with oliguria necessitated hemodialysis. A mild disseminated intravascular coagulation responded well to the administration of blood components. His neurologic status, attributed to a hypoxic insult, had improved to the point of withdrawal of all four limbs to deep painful stimuli, but "sedation paralysis" became necessary to control elevated intracranial pressure.

Thirty-six hours after admission to the Regional Trauma Unit, both right and left calves were firm to palpation. Twelve hours later both thighs were firm to palpation and both calves were becoming increasingly tight. The right dorsalis pedis and posterior tibial pulses were absent and the right foot felt cold. A four-compartment fasciotomy was performed on the calf of the right leg, using a medial and lateral approach. This restored pulses and Doppler measured flow to the right foot. A similar left

calf decompression procedure was performed 12 hours later.

On day 11 the fasciotomy sites were re-explored. Necrotic muscle in the anterior compartment of the right calf was debrided. Both limbs continued to be dressed daily. Fifteen days later, during a second fasciotomy revision and débridement, it was noted that necrotic muscle was present in all four compartments bilaterally, so below-knee amputations were performed the next day. Over the ensuing 4 weeks the patient had numerous problems with breakdown of skin at the stump sites and eventually required a left above-knee amputation and amputation through the right knee.

Discussion

This case demonstrates a serious complication resulting from the use of the pneumatic antishock garment — the development of bilateral lower extremity compartment syndromes, without evidence of coexisting trauma, necessitating bilateral lower limb amputations. It appears that prolonged hypotension combined with prolonged application of the PASG created severe compartment syndromes refractory to bilateral four-compartment fasciotomies. The patient's associated severe head injury may have masked the lower limb symptoms and contributed to late diagnosis and the poor outcome following compartment decompression.

In 1981, Maull and associates[8] described two patients who had compartment syndromes following lower extremity trauma and the use of the PASG. Both required amputation of the traumatized limb. In the same year, Johnson[15] described bilateral anterior tibial compartment syndromes in a 21-year-old man who suffered no obvious lower extremity trauma and in whom the PASG was inflated for 3.5 hours. This patient required resection of the entire anterior compartment and was left with a foot drop. Williams and colleagues[13] described another case of compartment syndromes without lower extremity trauma necessitating bilateral subcutaneous fasciotomies of the anterior and peroneal compartments. The patient was left with bilateral foot drop and sensory loss over both feet. More recently, Bass and associates[16] described two cases of thigh compartment syndromes in patients with prolonged (12 and 24 hours) use of the PASG without coexisting lower extremity trauma. Brotman and associates[14] also reported thigh compartment syndrome in a patient in whom the PASG had been improperly applied. In this case the abdominal compartment was left inflated following deflation of the legs and it was postulated that the abdominal compartment acted as a venous tourniquet. Godbout and colleagues[17] described a 20-year-old man, injured in a motor vehicle accident, who suffered necrosis of lower limb muscles bilaterally after application of the PASG for 180 minutes. Including our case, five of the nine reported cases of lower extremity compartment syndromes have occurred in association with prolonged inflation of the PASG and acute renal failure, indicative of prolonged hypotension.

A compartment syndrome is a condition in which increased pressure within an enclosed fascial space compromises the circulation and the function of tissues within that space.[18] Elevated tissue pressures will reduce the arterial–venous gradient, diminishing flow across the capillary bed. When local metabolic demands can no longer be met, tissue death ensues. Normally the arterial or precapillary pressures are approximately 25 mm Hg and postcapillary pressures are approximately 16 mm Hg. It is this arterial–venous gradient that establishes flow across the capillaries. Any factor that decreases the arterial head of pressure (shock) or increases the postcapillary venular pressure (venous obstruction, increased tissue pressure) will diminish the gradient and thus blood flow and oxygen delivery to the local tissues.[19] Experimental work suggests that when tissue pressures reach levels greater than 30 to 35 mm Hg, ischemic changes occur in nerve and muscle, and the reversibility of these changes will be determined by the amount and duration of increased tissue pressure.[20] It has recently been demonstrated[21] in healthy volunteers that more than 90% of the pressure supplied by the external use of the PASG is transmitted directly to the muscle compartment. Chisholm and Clark[21] performed experiments with healthy volunteers using Wick catheters inserted into the anterior tibial compartment for continuous pressure measurements. They demonstrated that, with inflation of the PASG to 60 mm Hg, pressures within the compartment greater than 60 mm Hg were consistently demonstrated. It should be noted that the muscle compartment pressure threshold for a compartment syndrome would be markedly reduced under conditions of prolonged hypotension.[22]

Although compartment syndromes have previously been described with the PASG, this case report demonstrates the possible attendant morbidity. The PASG should be used at the minimum inflation pressure necessary to maintain hemodynamic stability and removed as quickly as is safely possible. When prolonged use (longer than 1 or 2 hours) is necessary, close clinical examination, combined with compartment pressure measurements, when indicated, should be performed following removal of the PASG. In patients with head injuries or altered sensation, a high degree of suspicion is necessary to avoid missing a potentially serious condition.

References

1. CRILE GW: *Hemorrhage and Transfusion: Experimental and Clinical Research*, Appleton, New York, 1909
2. BIVINS HG, KNOPP R, TIERNAN C, et al: Blood volume displacement with inflation of antishock trousers. *Ann Emerg Med* 1982; 11: 409–412
3. GAFFNEY FA, THAL ER, TAYLOR WF, et al: Hemodynamic effects of Medical Anti-Shock Trousers (MAST garment). *J Trauma* 1981; 21: 931–937
4. GOLDSMITH SR: Comparative hemodynamic effects of antishock suit and volume expansion in normal human beings. *Ann Emerg Med* 1983; 12: 348–350
5. NIEMANN JT, STAPCZYNSKI JS, ROSBOROUGH JP, et al: Hemodynamic effects of pneumatic external counterpressure in canine hemorrhagic shock. *Ann Emerg Med* 1983; 12: 661–667
6. TRUNKEY DD: Is ALS necessary for pre-hospital trauma care? (E). *J Trauma* 1984; 24: 86–87
7. NIEMANN JT: Pneumatic anti-shock trousers: safety, benefit and physiological effect. *Ann Emerg Med* 1983; 12: 377
8. MAULL KI, CAPEHART JE, CARDEA JA, et al: Limb loss following Military Anti-Shock Trousers (MAST) application. *J Trauma* 1981; 21: 60–62
9. RANSOM KJ, MCSWAIN NE JR: Metabolic acidosis with pneumatic trousers in hypovolemic dogs. *JACEP* 1979; 8: 184–187
10. RANSOM K: Respiratory function following application of MAST trousers. *JACEP* 1978; 7: 297–299
11. WANGENSTEEN SL, DEHOLL JD, LUDEWIG RM, et al: The detrimental effect of the G-suit in hemorrhagic shock. *Ann Surg* 1969; 170: 187–192
12. SHENASKY JH II: The renal hemodynamic and functional effects of external counterpressure. *Surg Gynecol Obstet* 1972; 134: 253–258
13. WILLIAMS TM, KNOPP R, ELLYSON JH: Compartment syndrome after anti-shock trouser use without lower-extremity trauma. *J Trauma* 1982; 22: 595–597
14. BROTMAN S, BROWNER BD, COX EF: MAS trousers improperly applied causing a compartment syndrome in lower-extremity trauma. *Ibid:* 598–599
15. JOHNSON BE: Anterior tibial compartment syndrome following use of MAST suit. *Ann Emerg Med* 1981; 10: 209–210
16. BASS RR, ALLISON EJ JR, REINES HD, et al: Thigh compartment syndrome without lower extremity trauma following application of pneumatic antishock trousers. *Ann Emerg Med* 1983; 12: 382–384
17. GODBOUT B, BURCHARD KW, SLOTMAN GJ, et al: Crush syndrome with death following pneumatic antishock garment application. *J Trauma* 1984; 24: 1052–1056
18. MUBARAK SJ, HARGENS AR: Acute compartment syndromes. *Surg Clin North Am* 1983; 63: 539–565
19. MATSEN FA III, CLAWSON DK: The deep posterior compartmental syndrome of the leg. *J Bone Joint Surg [Am]* 1975; 57: 34–39
20. HARGENS AR, SCHMIDT DA, EVANS KL, et al: Quantitation of skeletal-muscle necrosis in a model compartment syndrome. *J Bone Joint Surg [Am]* 1981; 63: 631–636
21. CHISHOLM CD, CLARK DE: Effect of the pneumatic antishock garment on intramuscular pressure. *Ann Emerg Med* 1984; 13: 581–583
22. MUBARAK SJ, HARGENS AR: *Compartment Syndromes and Volkmann's Contracture*, Saunders, Philadelphia, 1981

BOOK REVIEWS

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include basic concepts, preoperative assessment (with particular emphasis on the cardiorespiratory system), anesthetic agents and equipment, and postoperative care. Areas of special expertise such as airway maintenance, intubation and resuscitation are all reviewed in appropriate detail for a book of this size. The actual administration of general anesthesia is covered in only 13 pages, but this is an adequate overview when considered in conjunction with the material covered elsewhere in the book. One chapter is devoted to patient care in the surgical intensive care unit. It outlines concisely the principles of intensive-care-unit management, including mechanical ventilation and its complications.

While principles of medical practice are essentially the same in all western countries, the details may differ, depending on long-

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A. FREIBERG, MD, FRCSC, FACS; C. HONG, MD

Apple-Coring Technique for Severe Gynecomastia

In evaluating cases of gynecomastia it is important to differentiate between physiologic and pathologic forms. The authors review gynecomastia with reference to its occurrence in various age groups, the pathologic features and etiology, and the differentiation and classification of the two types.

If the gynecomastia is large or long-standing, surgical treatment may be necessary. The authors review the various surgical techniques used from the early descriptions up to the present. The apple-coring technique has the advantages of leaving an inconspicuous scar, allowing correction of the hypertrophic areola and good chest-wall contour, maintaining the viability of the nipple and decreasing the common complications of inverted nipples and a doughnut-shaped deformity. The results and outcome in four patients treated by the apple-coring technique are reported. Although the method is not perfect, the results are encouraging and the technique is an addition to the armamentarium of surgeons treating large gynecomastia.

Lorsqu'on procède à l'évaluation d'un cas de gynécomastie, il est important de faire la différence entre les formes physiologiques et pathologiques de la maladie. Les auteurs étudient divers aspects de la maladie dont son apparition dans les différents groupes d'âge, les caractéristiques pathologiques et l'étiologie, de même que la différenciation et la classification des deux types.

Il peut être nécessaire d'opérer si la gynécomastie est importante ou présente de longue date. Les auteurs passent en revue les diverses techniques chirurgicales utilisées, depuis les premières descriptions jusqu'à nos jours. La technique rappelant l'enlèvement d'un trognon de pomme a les avantages de laisser une cicatrice discrète, de permettre la correction d'une aréole hypertrophique et du contour thoracique, de préserver la viabilité du mamelon et de réduire les complications fréquentes que représentent l'inversion du mamelon ou une difformité en forme de beignet. Les résultats obtenus chez quatre patients traités avec cette technique sont rapportés. Bien que la méthode soit imparfaite, les résultats sont encourageants et la technique s'ajoute aux moyens dont disposent les chirurgiens traitant les gynécomasties importantes.

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Gynecomastia — unilateral or bilateral benign enlargement of the male breast — may take the form of either a discrete, palpable subareolar plate of mammary tissue, easily distinguished from adipose tissue, or a more diffuse mass that resembles surrounding fat.

Minimal enlargement can easily be corrected surgically by various well-accepted techniques with good cosmetic results, but the larger, female-type breast, presents a problem and a surgical challenge. A large amount of breast tissue must be removed without leaving external scars. A popular method is the apple-coring technique described by Huang and associates[1] in 1982. They reported on 24 patients with excellent results.

In this paper we review the topic of gynecomastia and describe our early experiences in four men with large gynecomastia using the apple-coring technique.

Although this technique is not perfect, the results to date are encouraging and provide the surgeon with a valuable addition to the surgical armamentarium.

Occurrence of Gynecomastia

Asymptomatic gynecomastia has been reported in 30% to 39% of asymptomatic healthy males who are not on any medication. Nuttall[2] reviewed the prevalence in different age groups and found three periods of life during which there is a rapid increase in rate of occurrence — puberty (age 12 to 15 years), early adult life (age 19 to 25 years) and middle age, confirming an earlier autopsy study by Williams[3] who reported a rate of around 40%.

Pathological Features

The histologic appearance depends on the duration of the developmental process. In early puberty, gynecomastia typically shows prominent ductules embedded in loose connective tissue. Gynecomastia of several years' duration usually appears as inconspicuous ductular structures in a dense, hyalinized and fibrous stroma. Resolution occurs by reduction in duct size and epithelial content with gradual disappearance of the ducts, leaving hyaline bands that eventually disappear.

Etiologic Aspects

A central issue in evaluating cases of gynecomastia is the differentiation between physiologic and pathologic.

The growth of the male breast, as in females, is mediated by the trophic and stimulatory effect of estrogens on mammary epithelial tissues. Androgens have an inhibitory action on the mammary epithelial tissues. Gynecomastia may, therefore, result from an imbalance between these two factors. The role of prolactin, proven to promote lactation, in the genesis of gynecomastia is unclear.

Serum prolactin levels are normal in most patients with gynecomastia and enlarged breasts seldom develop in those with hyperprolactinemia.

Physiologic Gynecomastia

This type of gynecomastia occurs in three different age groups.

• Newborn. Transient enlargement results from the actions of maternal or placental estrogens, or both. The enlargement ordinarily disappears in a few weeks, although it has been known to persist longer.

• Adolescent. Gynecomastia in this age group is very common. The median age of onset is 14 years; it is often asymmetric. The onset correlates with transient elevations of plasma estradiol levels before puberty is complete.

• Elderly. At least 40% of elderly men have gynecomastia, possibly resulting from an increased peripheral conversion of androgens to estrogens. However, the more frequent occurrence of abnormal liver function and of drug therapy may account for this, in which case the gynecomastia would be classified as pathologic.

Pathologic Gynecomastia

Three basic mechanisms account for the pathologic type of gynecomastia.

• Deficiency in testosterone production or action.

• Increase in estrogen production.

• Drugs that act through the above two mechanisms or by mechanisms that have not been defined (Table I).

Diagnosis

Since treatment depends partially on the etiology, it is important to differentiate between physiologic and pathologic causes.

A careful history should include patient age, time of onset of the gynecomastia, family history, endocrinologic functions such as libido, fertility and amount of body hair and any medication the patient is taking.

Physical examination must be thorough, with special emphasis on factors that may indicate the effects of androgen (testicular size, liver size, body shape).

Laboratory investigation should complement the history and physical examination. This may include liver function studies and measurement of endocrine levels (24-hour urinary 17-ketosterone, plasma estradiol, luteinizing hormone and testosterone). Special investigations, such as karyotyping and bronchoscopy may be necessary as indicated by the history and physical examination.

Surgical Treatment

When the primary cause of gynecomastia is identified and corrected early, the breast enlargement usually subsides promptly. If gynecomastia is large or of long duration, surgical treatment may be necessary.

There are three important considerations in the surgical treatment of gynecomastia — the amount of excessive breast or adipose tissue, a hypertrophic nipple-areolar complex and the amount of redundant skin. Thus, gynecomastia can be practically classified as shown in Table II.

According to Simon and associates,[4] grades 1 and 2a are the most common in adolescence and usually subside spontaneously. Grades 2b and 3 are more common in the middle aged and elderly. They are usually of long standing or occur when there has been marked weight loss.

Table I—Causes of Gynecomastia

Increased estrogen production
Increased secretion
Hermaphroditism
Klinefelter's syndrome
Congenital adrenal hyperplasia
Neoplastic
Adrenal carcinoma
Testicular tumour (Sertoli cell, Leydig cell, choriocarcinoma)
Neoplasm secreting human chorionic gonadotrophin (lung, liver, kidney, stomach, lymphopoietic system)
Increased peripheral conversion (peripheral aromatase)
Adrenal disease
Liver disease
Starvation
Thyrotoxicosis
Drug related
Oral contraceptives, digitalis, marihuana, heroin, estrogens
Deficient production or action of testosterone
Decreased production
Congenital anorchia
Klinefelter's syndrome
Infectious orchitis
Panhypopituitarism
Castration
Paraplegia
Muscular dystrophy
Renal failure
Androgen resistance due to receptor protein abnormalities
Testicular feminization
Reifenstein syndrome
Pharmacologic
Cimetidine, spironolactone, alkylating agent
Idiopathic

Table II—Classification of Gynecomastia

A	1	Unilateral
	2	Bilateral
B		Nipple-areolar complex
	1	< 2 cm diameter
	2	> 2 cm diameter
C		Grades (skin and amount of breast enlargement)
	1	Small visible breast enlargement without skin redundancy
	2a	Moderate breast enlargement without skin redundancy
	2b	Moderate breast enlargement with skin redundancy
	3	Marked breast enlargement with marked skin redundancy (i.e., pendulous breast)

Operative Technique

The first report of mastectomy for gynecomastia dates back to the seventh century by Paulus Aegineta,[5] who used a submammary lunar incision. In the last 100 years, there have been many refinements in the surgical technique (Table III[1,4-17]). We used a circumareolar

Table III—Surgical Technique Used for Gynecomastia

Series	Technique/Approach
Aegineta, 7th century[5]	Submammary lunar
Dufourmentel, 1928[6]	Inferior semicircular intra-areolar
Kurtzahn, 1928[7]	Intra-areolar and upward transposition
Vogt, 1941[8]	Skin excision and transposition
Campos, 1942[9]	Skin excision and transposition
Webster, 1946[10]	Inferior semicircular intra-areolar
Malbec, 1946[11]	Skin excision and transposition
Pitanguy, 1966[12]	Intra-areolar bisecting nipple and areola
Letterman and Schurter, 1969[13]	Superior semicircular intra-areolar
Letterman and Schurter, 1972[14]	Oblique mammaplasty
Simon and associates, 1973[4]	Inverted omega
Wray and associates, 1974[15]	Radial mastectomy and free graft of nipple and areola
Davidson, 1979[16]	Concentric circular
Huang and associates, 1982[1]	Circumareolar
Saad, 1983[17]	Circumareolar

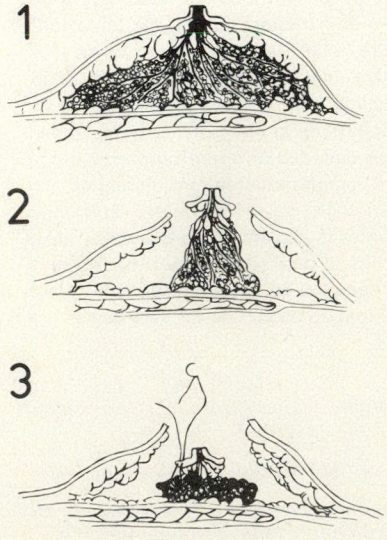

FIG. 1—Diagrammatic illustration of apple-coring technique.

approach (the apple-coring technique) in four patients (Fig. 1).

First the breast mound is outlined to determine the amount of tissue to be excised. An incision is made through the full thickness of the areolar skin to expose the underlying breast tissue (Fig. 2). The skin overlying the breast mound is then dissected in a centrifugal manner to the limits of the breast mound as marked (Figs. 3 and 4). The apple-coring technique is used to remove excess breast tissue. The surgeon must avoid excising too much tissue which would create a doughnut-shaped deformity and compromise the vascular supply to the nipple. The nipple is then imbricated with absorbable sutures to decrease the nipple height and to obliterate the dead space. Nonabsorbable sutures are used for skin closure. Liposuction may be added to feather the periphery and minimize any doughnut-shaped deformity. Hemovac suction drains are used for 4 to 5 days.

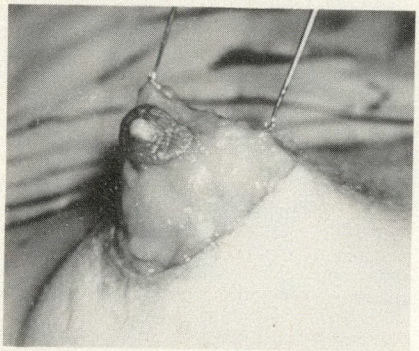

FIG. 2—Development of nipple-areola pedicle (apple core).

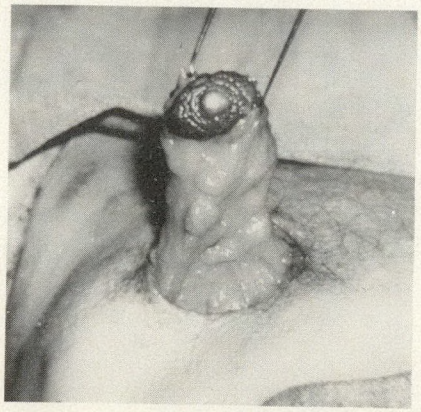

FIG. 4—Final appearance of pedicle.

A compressive adhesive dressing is left in place for another 2 to 3 days. The periareolar sutures are removed 7 to 10 days postoperatively.

Patients and Results (Table IV)

We operated on four patients with large gynecomastia using this technique. One patient (case 4) had minor hematomas in both breasts, but drainage was not required.

Marginal areolar necrosis caused delayed healing in five breasts (Fig. 5). One patient (case 1) had partial areolar necrosis in both breasts that took approximately 6 weeks to heal completely. The final results were satisfactory (Fig. 6).

Follow-up ranged from 3 to 18 months. Two patients were very happy with the results, the other two said they were "quite happy" (Figs. 6 and 7).

Liposuction, in an attempt to feather the periphery, was used in one patient but was only partially successful because only very small amounts of fat could be removed by this technique.

The resulting scars were acceptable to both the patient and the surgeon.

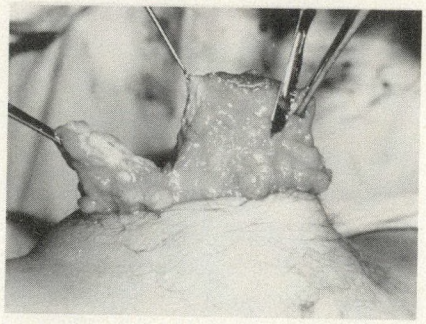

FIG. 3—Trimming of pedicle in centrifugal manner.

FIG. 5—Appearance of periareolar scars after minor marginal areolar necrosis of left breast in case 4, 6 months after operation.

Table IV—Summary of Four Cases of Gynecomastia Managed Surgically by the Apple-Coring Technique

Patient no.	Age, yr	Weight of tissue removed, g	Complications: Hematoma Right	Hematoma Left	Marginal areolar necrosis Right	Marginal areolar necrosis Left	Follow-up, mo	Subjective results
1	15	400	0	0	Minor	Moderate	8	Quite happy
2	27	240	0	0	0	Minor	3	Quite happy
3	32	125	0	0	0	Minor	6	Very happy
4	41	197	Minor	Minor	0	Minor	18	Very happy

Discussion of Other Surgical Techniques

Various techniques have been used in the surgical correction of gynecomastia. They can be classified into intra-areolar and extra-areolar.

With the standard submammary approach, good exposure is achieved for the dissection of the breast tissue with possible reduction of excessive skin as well. The problem with this technique is the obvious scarring and downward migration of the nipples if too much skin is excised. Other extra-areolar incisions used to correct the skin redundancy were modified techniques, originally designed for reduction of the female breast. The resultant scar thus usually consists of an inconspicuous periareolar portion plus a more prominent medial or, more commonly, a lateral oblique component or the classic inverted-T component.

The disadvantages of the Webster or Dufourmentel semicircular, periareolar approach are poor exposure for dissection and the problem of redundant skin. With poor exposure, postoperative hematoma is a serious complication. The large area of dead space created may predispose to seroma formation.

The circumareolar approach combined with concentric excision of redundant skin or areola, or both, has the advantages of a relatively inconspicuous scar (Fig. 5), correction of the hypertrophic areola and a potential for good chest-wall contouring (Figs. 6 and 7). Nipple viability is based on the fact that the nipple-areolar complex derives its blood supply from the perpendicular perforators of the third, fourth and fifth intercostal arteries which are often 1 to 2 mm in diameter.

The fourth interspace perforator is the principal artery and can maintain the viability of the nipple-areolar complex. (The other significant arterial supply to the breast is the internal and external mammary vessels coming from the medial and lateral sides respectively.) With proper contouring of the chest wall, leaving at least 1.5 cm of breast tissue over the pectorial fascia, imbricating the nipple stalk and adding liposuction, one can decrease the common complications of nipple inversion and the doughnut-shaped deformity. Partial nipple necrosis is a potential complication with this procedure.

References

1. HUANG TT, HIDALGO JE, LEWIS SR: A circumareolar approach in surgical management of gynecomastia. *Plast Reconstr Surg* 1982; 69: 35–40

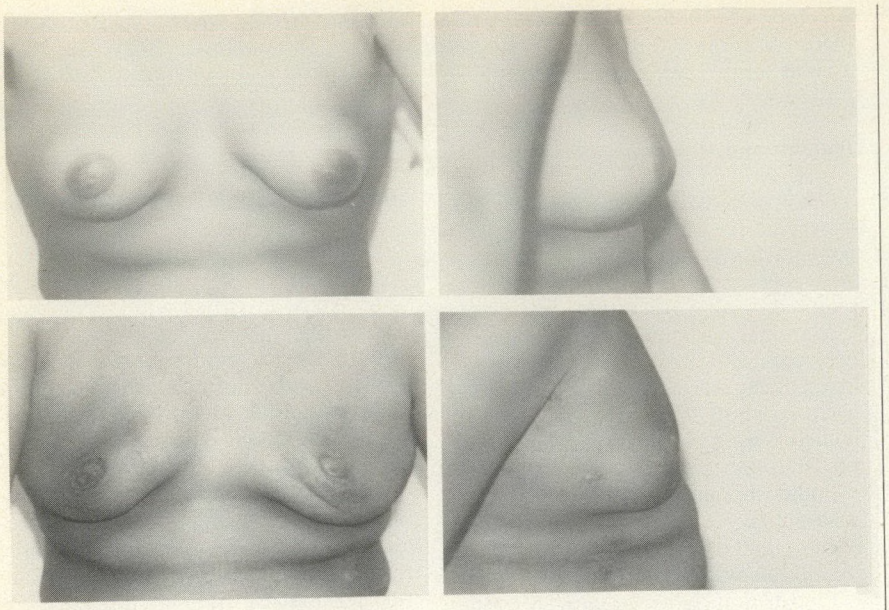

FIG. 6—Case 1, 15-year-old boy with gynecomastia. Appearance (top) preoperatively and (bottom) 8 months postoperatively.

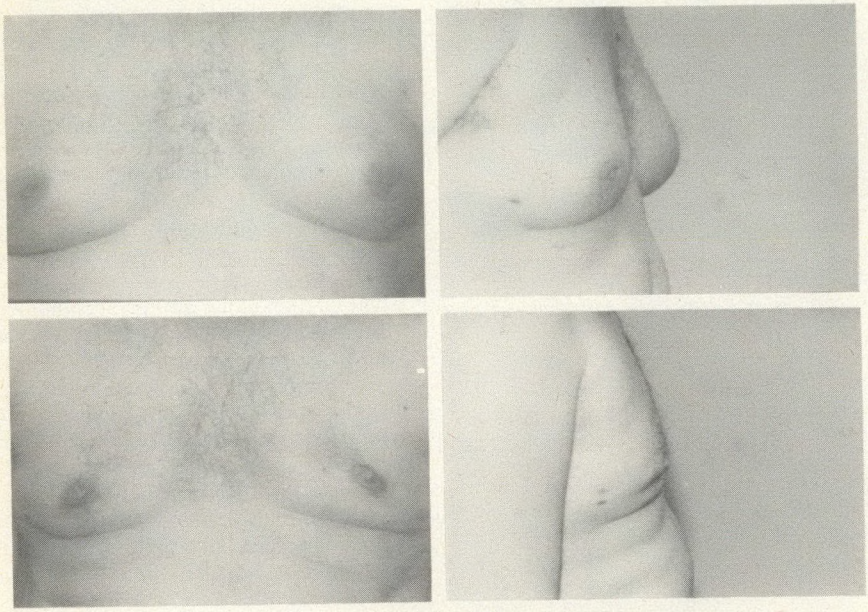

FIG. 7—Case 4. Appearance (top) preoperatively and (bottom) 10 months postoperatively.

2. NUTTALL FQ: Gynecomastia as a physical finding in normal men. *J Clin Endocrinol Metab* 1979; 48: 338–340

3. WILLIAMS MJ: Gynecomastia. Its incidence, recognition, and host characterization in 447 autopsy cases. *Am J Med* 1963; 34: 103–112

4. SIMON BE, HOFFMAN S, KAHN S: Classification and surgical correction of gynecomastia. *Plast Reconstr Surg* 1973; 51: 48–52

5. AEGINETA P: *The Seven Books of Paulus Aegineta,* vol 2, bk 4, Syndenham Society, London, 1847: 344

6. DUFOURMENTEL L: L'incision aréolaire dans la chirurgie du sein. *Bull Mem Soc Chir* 1928; 20: 8–14

7. KURTZAHN H: Zur Operation der Gynäkomastie und der Hangeburst. *Dtsch Z Chir* 1928; 209: 403–406

8. VOGT LG: Beitrag zur plastichen Operation der gynäkomastie. *Chirurg* 1941; 13: 322–324

9. CAMPOS F: Sobre um caso de ginecomastia bilateral e seu tratamento cirurgico. *Arq Cir Clin Exp* 1942; 6: 703–705

10. WEBSTER JP: Mastectomy for gynecomastia through a semi-circular intra-areolar incision. *Am Surg* 1946; 124: 557–575

11. MALBEC EF: Cirurgia plastic: ginecomastia: tecnica operatoria. *Dia Med* 1946; 18: 375–376

12. PITANGUY I: Transareolar incision for gynecomastia. *Plast Reconstr Surg* 1966; 38: 414–419

13. LETTERMAN G, SCHURTER M: The surgical correction of gynecomastia. *Am Surg* 1969; 35: 322–325

14. Idem: Surgical correction os massive-gynecomastia. *Plast Reconstr Surg* 1972; 49: 2359–2362

15. WRAY RC JR, HOOPES JE, DAVIS GM: Correction of extreme gynaecomastia. *Br J Plast Surg* 1974; 27: 39–41

16. DAVIDSON BA: Concentric circle operation for massive gynecomastia to excise the redundant skin. *Plast Reconstr Surg* 1979; 63: 350–354

17. SAAD MN: An extended circumareolar incision for breast augmentation and gynecomastia. *Aesthetic Plast Surg* 1983; 7: 127–128

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Craniocervical Necrotizing Fasciitis: Critical Factors in Management

Necrotizing fasciitis involving the head and neck is rare. The authors describe two such patients treated at their institution and analyse 39 cases reported in the literature. This entity may be divided into two groups based on the site of origin of the infection: group 1 (13 cases) infections, originating in the scalp and eyelids, mostly secondary to trauma, do not progress rapidly, respond well to medical and operative measures and result in minimal permanent disability. These infections usually are caused by hemolytic streptococci and *Staphylococcus aureus.* Group 2 (28 cases) infections, originating in the face or neck and mostly complications of dental and pharyngeal sepsis, progress rapidly to adjoining sites including the chest wall and mediastinum. These infections are caused by a wide variety of microorganisms including anaerobes; fatal complications are frequent and the death rate is high (32%). Early and very aggressive débridement and drainage are mandatory and should be repeated if warranted.

La fasciite nécrosante de la tête et du cou est rare. Les auteurs en décrivent deux cas qui furent traités dans leur établissement et analysent 39 cas signalés dans la littérature. On peut distinguer deux groupes selon le foyer d'origine de l'infection: les infections du groupe 1 (13 cas) qui originent du cuir chevelu et des paupières et qui sont le plus souvent secondaires à un traumatisme, ne progressent pas rapidement, répondent bien aux traitements médical et chirurgical et laissent une incapacité permanente réduite. Ces infections sont habituellement dues à un streptocoque hémolytique et à *Staphylococcus aureus.* Les infections du groupe 2 (28 cas) originent du visage ou du cou, sont la plupart du temps une complication d'une infection dentaire ou pharyngée et progressent rapidement vers des structures adjacentes comme la paroi thoracique et le médiastin. Ces infections sont le fait d'un large spectre de microorganismes, dont des anaérobies; les complications fatales sont fréquentes, la mortalité est élevée (32%). Un débridement précoce et très agressif et ainsi qu'un drainage sont obligatoires; la procédure doit être répétée si nécessaire.

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Necrotizing fasciitis is a relatively uncommon but severe soft-tissue infection characterized by extensive necrosis of the superficial fascia with widespread undermining of the surrounding tissues. When diagnosis and treatment are delayed, morbidity and mortality are alarming. This entity was described in the earlier literature as "hospital gangrene"[1] and "streptococcal gangrene".[2] The term "necrotizing fasciitis" was first used by Wilson[3] because it emphasized the pathogenesis of the lesion. The most commonly affected areas are the abdomen, perineum and extremities and, rarely, the head and neck. We report two cases of necrotizing fasciitis of the neck to emphasize the critical factors in diagnosis and management of this relatively rare entity.

Case Reports

Case 1

A 43-year-old man was admitted to the otolaryngology service with a 1-year history of "sore throat" and history of dysphagia with constant pain in the right side of the face and neck for a few weeks. He drank and smoked heavily and had lost 14 kg. A large fungating lesion involving the base of the tongue, right tonsil, retrotonsillar area and lateral pharyngeal wall was noted on examination. Firm, tender, enlarged cervical nodes were palpable bilaterally, giving the impression of a large oropharyngeal carcinoma with cervical node involvement. The hemoglobin level, leukocyte count and chest x-ray film were all normal.

Two days after admission, fever developed (temperature 40°C) with a productive cough and mild respiratory distress. The right neck, clavicular area and anterior chest wall were noted to be erythematous, indurated and tender. The leukocyte count increased to 20.0×10^9/L and the chest film revealed a right lower lobe pneumonia. Cephalothin (1 g every 6 hours) was given intravenously. Biopsies of the intraoral lesion revealed infiltrative squamous cell carcinoma of the tongue. X-ray films of the neck showed partial airway obstruction due to the right neck swelling. On repeat chest roentgenography there was a persistent pneumonia and a right pleural effusion. The antibiotic coverage was changed to tobramycin (80 mg every 8 hours) and ampicillin (1 g every 6 hours).

On hospital day 5 the elevated temperature persisted, the induration and swelling in the neck extended across the midline of the chest and was accompanied by crepitation. Necrotizing fasciitis initiated by necrosis of the oropharyngeal lesion was suspected. Panendoscopy and surgical exploration of the neck were performed. There was extensive necrosis of the fascia and muscles of the right side of the neck, the process crossing the midline anteriorly and extending into the superior mediastinum. Proximally the necrosis involved the oral cavity, through the parapharyngeal space. Multiple collections of foul-smelling pus were drained and devitalized tissue debrided radically with the overlying skin. The neck spaces and the mediastinum were extensively drained. Cultures grew *Staphylococcus epidermidis* and *Streptococcus faecalis.* Anaerobic cultures isolated *Micrococcus luteus.* The antibiotic coverage was changed to massive doses of penicillin (2 million units every 2 hours) together with tobramycin and clindamycin.

Initially, the patient's general condition improved, but additional necrosis of tissue inferior to the clavicle was noted 3 days after antibiotic coverage was changed, with persistent purulent drainage from the mediastinum. Wider excision of necrotic tissue over and below the clavicles and on the anterior chest wall was performed. A collection of purulent material on the right lateral chest wall over the sixth and seventh ribs, communicating with the infraclavicular necrotic process, was drained extensively with counterincisions. The open areas extended to the posterior triangles of the neck bilaterally, from the submandibular areas to the mid-sternum and from the lateral end of the clavicle to the mid-clavicle on the left (Fig. 1).

Follow-up x-ray films revealed a persistent loculated pleural effusion in the right upper thorax. Under the guidance of computerized tomography this loculation was drained percutaneously. Increasing left pleural effusion

developed and failed to resolve after thoracentesis, necessitating a left thoracotomy and decortication on the 40th hospital day. He recovered from this procedure and was successfully weaned from the ventilator. The neck and chest wounds continued to heal satisfactorily and split-thickness skin grafts were eventually applied over the granulating areas. As the intraoral lesion was deemed inoperable, radiotherapy was initiated while he was in the hospital and he was transferred to the otolaryngology service for further care.

Case 2

A 31-year-old woman sustained blunt trauma to the face resulting in a fracture of the left mandible near its angle. She sought medical help 1 week later and the fracture was treated by closed reduction at another hospital. She failed to keep her follow-up appointment and during the next 2 weeks noticed increasing swelling, erythema and pain over the left side of the jaw, extending into the neck. A black, discoloured patch appeared in the skin over the fracture site and discharge of purulent material from this area prompted her to visit our emergency department. She looked ill and had a temperature of 38.5°C. Gangrene of the skin over the angle of the mandible was evident, with erythema, induration and tenderness in the submandibular and submental areas and also in the upper left side of the neck (Fig. 2). She had a history of hypertension and alcoholism for several years. X-ray films revealed the mandibular fracture, but no gas was noted in the soft tissues. Results of laboratory studies were normal except for leukocytosis (13.5×10^9/L).

Immediately after admission to hospital the lower left third molar, which was in line with the fracture, was extracted with débridement of the devitalized tissue in the submandibular submental areas, using wide drainage. The necrotic tissues contained foul smelling pus which grew *S. aureus, Clostridium perfringens, Bacteroides fragilis* and anaerobic diphtheroids. She was treated intravenously with gentamicin (80 mg every 8 hours), clindamycin (600 mg every 6 hours) and penicillin (2 million units every 4 hours) from the time of admission. The area of the fracture was left open for periodic irrigations with saline solution and for frequent dressing changes. However, copious yellow purulent drainage from the wound persisted and she continued to be febrile. With evidence of infection spreading down the left side of the neck, exploration on hospital day 4 revealed extension of the necrotizing process down to the supraclavicular area and posteriorly to the trapezius muscle. Radical débridement of necrotic tissue and overlying skin was accomplished with evacuation of all loculated purulent collections. Multiple irrigation and suction catheters were placed in the dependent areas.

With saline wound irrigation and frequent minor bedside débridement, her general condition improved significantly and the wounds began to heal. She received nutritional support by continuous feeding through a nasogastric tube. The irrigation catheters were removed 5 days after the second procedure. The open fracture was soon covered with granulation tissue and 17 days after admission split-thickness skin grafts were applied over the open wounds. She was discharged on day 24 with good functional and cosmetic results.

Discussion

An extensive review of the literature since 1945 yielded 41 cases of necrotizing fasciitis of the head and neck (Table I[4–32]). There were two previous reviews of the literature, but one report,[20] with a review of eight cases omitted certain pertinent data necessary for accurate evaluation.[20] The second report attempted to classify the 32 cases based on bacterial cultures.[30] Neither report alluded to the site of origin of the infection as a critical factor in the final outcome. We classified these 41 cases into two groups: infections originating in the eyelids or scalp and infections originating in the face or neck. There are significant differences in the virulence of infections, sites of extension, complications and prognosis between the two groups (Table II).

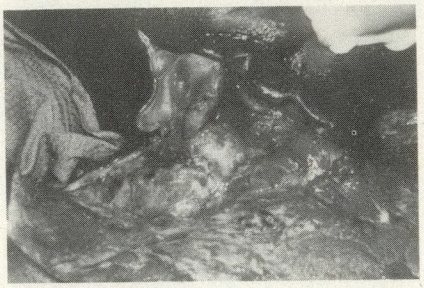

FIG. 1—Case 1. Extensive cervical necrotizing fasciitis after débridement. Wound extends down anterior chest wall, inferior to clavicles and laterally to trapezius muscles.

FIG. 2—Case 2. Necrotizing fasciitis originating over fractured mandible and extending down left side of neck.

Group 1

Infections originating in the scalp.— Scalp involvement was observed in six patients (Table I). Blunt and penetrating trauma were the etiologic factors in all of them and coincidentally four of the six patients also had diabetes mellitus. Infec-

Table I—Craniocervical Necrotizing Infections — Reported Cases

Authors	No. of cases
Infections originating in scalp	
Moore and associates, 1950[4]	2
Argamaso, 1966[5]	1
Fisher and associates, 1979[6]	1
Skef and associates, 1981[7]	2
Infections orginating in eyelids	
Mortada, 1964[8]	1
Schott, 1966[9]	1
Beathard and Guckian, 1967[10]	1
Buchanan, 1970[11]	1
Ross and Kohlhepp, 1973[12]	2
Carruthers and associates, 1975[13]	1
Infections originating in face and neck	
Collins and Nadel, 1965[14]	1
Stone and Martin,* 1972[15]	2
Cogan, 1973[16]	1
Crowson, 1973[17]	1
Richardson and associates, 1975[18]	1
Roser and associates, 1977[19]	1
Bahna and Canalis, 1980[20]	1
Mruthyunjaya, 1981[21]	1
Krespi and associates, 1981[22]	2
Gallia and Johnson, 1981[23]	1
Wills and Vernon, 1981[24]	3
Thompson and associates, 1982[25]	1
Ward and Berry, 1982[26]	1
Klau and Johnson, 1982[27]	1
Drake-Lee and associates, 1983[28]	1
Wenig and associates, 1984[29]	2
Spankus and associates, 1984[30]	3
Bush and associates, 1984[31]	1
Pacz and Czappan, 1984[32]	1
Present report, 1986	2
Total	41

*Data incomplete.

Table II—Comparison of Clinical Data

Factors	Infections originating in Scalp and eyelids, no. (%) (n = 13)	Infections originating in Face and neck, no. (%) (n = 26)
Etiology: Infection	3 (23)	20 (77)
Trauma	8 (62)	2 (8)
Infecting organisms	Hemolytic streptococci *Staphylococcus aureus*	Streptococci, staphylococci, anaerobes, gram-negative organisms
Thoracic extensions of infection	0	17 (65)
Other fatal complications	0	6*(23)
Mortality	0	7 (27)

*Tracheal compression (3), carotid occlusion (1), meningitis (1), rapid spread to abdomen and thigh (1).

tion extended to the entire scalp, ears, eyelids and neck in all cases but there were no deaths in this subgroup. All patients responded well to surgical management which consisted of débridement, drainage and irrigation.

Infections originating in the eyelids.—There were seven reported cases in which the origin of infection was the eyelids (Table I). The age of these patients ranged from 43 to 93 years. The most common cause of infection was trauma (four cases) followed by eyelid infection (two cases) and pruritis (one case). Extension of infection to adjoining sites, limited to the nose, cheek or face was recorded in three of the seven patients. In none of the cases was there involvement of the neck or chest. The surgical management of these patients consisted of débridement and drainage; split-thickness skin grafting was required in three. Recovery was smooth in all and no patient suffered permanent loss of vision. The only death in this group was from a totally unrelated abdominal catastrophe[12] (perforated duodenal ulcer proven by autopsy). The only organisms cultured in the 13 group 1 patients were *S. aureus* and hemolytic streptococci.

Group 2

Infections originating in face and neck.—Infections in the second and the larger group of 28 patients, including our two cases, originated in the face or neck and extended, to a varying degree, along the tissue planes of the neck (Table I). For a better understanding of this process, a brief outline of the anatomy of the cervical fascia, as exemplified by Levitt,[33] is appropriate.

The cervical fascia consists of the superficial fascia and three layers of deep fascia. The superficial fascia resembles subcutaneous tissue elsewhere in the body, but it also contains voluntary muscle in its deep positions — the platysma in the neck and the muscles of expression in the face. A potential space separates this from the deep cervical fascia. The deep cervical fascia is divided into three layers — superficial, middle and deep. These layers are not histologically different and actually are continuous with each other. All three layers contribute to the carotid sheath and an infection of any layer of the deep fascia may spread directly to the carotid sheath. The carotid sheath extends from the base of the skull to the upper mediastinum and permits unobstructed and uncontrolled spread of cervical infections into the chest.

Clinical information for analysis was complete in 26 of the 28 patients. The etiologic factors varied but dental infection was the single most common source (11 of 25 cases) followed by tonsillar (3 cases) and pharyngeal (3 cases) infections. In both of our patients the etiology was uncommon. Tumour necrosis leading to necrotizing fasciitis of the neck and chest has not been reported previously. Though soft-tissue trauma was reported as the cause in one patient,[20] fracture of facial bones as in our second case has not been recorded. Associated diseases or illnesses were neither consistent nor uniform and could not be correlated with the onset and severity of infection or the outcome in these 26 patients. Alcoholism (five patients) and diabetes mellitus (four patients) were the most common associated illnesses.

Compared with the patients with infections of the scalp and eyelids, the hospital course of the patients in this group was marked by rapid progression of fascial necrosis leading to fatal complications in some. Despite aggressive surgical débridement and adequate antibiotic therapy, the necrosis extended into the chest wall and mediastinum in 65% of the cases. All the reported fatalities in craniocervical necrotizing fasciitis belonged to this group (Table II).

Interestingly, there was direct communication with the oral cavity or the oropharynx from the site of origin of the infection in all these patients. The abundance of both aerobic and anaerobic organisms in the oropharynx contributes to the virulence of the spreading necrosis. This is exemplified by the isolation of several types of microorganisms, both aerobic and anaerobic, in the drainage material. While the initial cultures grew organisms that are common pathogens of the oropharynx, gram-negative nosocomial organims such as *Enterobacter* and *Pseudomonas* were isolated in 7 of the 25 patients after 1 week of hospitalization.

The death rate was 32% (9 of 28 patients) in the group. A comparison of survivors and nonsurvivors reveal them to be in the same age group and share the same etiologic factors. The major differences were the presence of associated illness, mainly diabetes and alcoholism (71% in nonsurvivors and 26% in survivors) and the presence of thoracic involvement by the infection (85% versus 57%). Other complications in the nonsurvivors — acute airway obstruction on admission (three cases) and very rapid extension of necrosis down to the flank and thigh (one case) — also contributed to the fatal outcome.

Early and aggressive surgical débridement of the necrotic tissue really is the only effective way to control the progression of the disease. Failure to achieve satisfactory drainage and débridement results in further spread of the necrotizing process in adjoining areas, as evidenced by thoracic extension of the infection in nearly two-thirds of the cases.

Thoracic involvement varies from necrosis of the fascia of the chest wall to mediastinitis, pleural effusion, empyema and even pericardial effusion. One of our patients manifested all the signs of thoracic extension of the infection necessitating multiple surgical procedures (mediastinal drainage, thoracotomy and decortication).

References

1. BROOKS SM: *Civil War Medicine,* Thomas, Springfield, Ill., 1966: 84
2. MELENEY F: Hemolytic streptococcal gangrene. *AMA Arch Surg* 1924; 9: 317–364
3. WILSON B: Necrotizing fasciitis. *Ann Surg* 1952; 18: 416–431
4. MOORE JR, GERRIE J, ELLIOTT H: Massive cellulitis of the scalp in persons with diabetes. *AMA Arch Surg* 1950; 60: 897–905
5. ARGAMASO RV: Synergistic gangrene: case reports. *Plast Reconstr Surg* 1966; 38: 16–22
6. FISHER JR, CONWAY MJ, TAKESHITA RT, et al: Necrotizing fasciitis. Importance of roentgenographic studies for soft-tissue gas. *JAMA* 1979; 241: 803–806
7. SKEF Z, HARDING R, GRAHAM WP III: Disseminated necrotizing fasciitis of the scalp. *Ann Plast Surg* 1981; 6: 322–326
8. MORTADA A: Post operative gangrene of eyelid. *Br J Ophthalmol* 1964; 48: 114–117
9. SCHOTT EG: Gangrene of the eyelids. *Ind Med Surg* 1966; 35: 27–29
10. BEATHARD GA, GUCKIAN JC: Necrotizing fasciitis due to group A beta-hemolytic streptococci. *Arch Intern Med* 1967; 120: 63–67
11. BUCHANAN CS: Necrotizing fasciitis due to group A beta-hemolytic streptococci. *Arch Dermatol* 1970; 101: 664–668
12. ROSS J, KOHLHEPP PA: Gangrene of the eyelids. *Ann Ophthalmol* 1973; 5: 84–88
13. CARRUTHERS A, CARRUTHERS J, WRIGHT P: Necrotizing fasciitis with polymyositis. *Br Med J* 1975; 3: 355–356
14. COLLINS RN, NADEL MS: Gangrene due to the hemolytic streptococcus — a rare but treatable disease. *N Engl J Med* 1965; 272: 578–580
15. STONE HH, MARTIN JD JR: Synergistic necrotizing cellulitis. *Ann Surg* 1972; 175: 702–711
16. COGAN IC: Necrotizing mediastinitis secondary to descending cervical cellulitis. *Oral Surg Oral Med Oral Pathol* 1973; 36: 307–320
17. CROWSON WN: Fatal necrotizing fasciitis developing after tooth extraction. *Am Surg* 1973; 39: 525–527
18. RICHARDSON JD, FOX GL, GROVER FL, et al: Necrotizing fasciitis complicating dental extraction (C). *Arch Surg* 1975; 110: 129
19. ROSER SM, CHOW AW, BRADY FA: Necrotizing fasciitis. *J Oral Surg* 1977; 35: 730–732
20. BAHNA M, CANALIS RF: Necrotizing fasciitis. (Streptococcal gangrene) of the face. Report of a case and review of the literature. *Arch Otolaryngol* 1980; 106: 648–651
21. MRUTHYUNJAYA B: Necrotizing fasciitis: report of case. *J Oral Surg* 1981; 39: 60–62
22. KRESPI YP, LAWSON W, BLAUGRUND SM, et al: Massive necrotizing infections of the neck. *Head Neck Surg* 1981; 3: 475–481
23. GALLIA LJ, JOHNSON JT: Cervical necrotizing fasciitis. *Otolaryngol Head Neck Surg* 1981; 89: 935–937
24. WILLS PI, VERNON RP JR: Complications of space infections of the head and neck. *Laryngoscope* 1981; 91: 1129–1136
25. THOMPSON JW, COLMAN MF, ZIMMERMAN M, et al: Necrotizing fasciitis of the neck. *Ear Nose Throat J* 1982; 61: 376–378
26. WARD NO, BERRY DW: Necrotizing fasciitis of the head and neck. *Ariz Med* 1982; 39: 390–393
27. KLAU M, JOHNSON JT: Cervical necrotizing fasciitis. *Trans Pa Acad Ophthalmol Otolaryngol* 1982; 35: 160–165
28. DRAKE-LEE AB, BROUGHTON SJ, RAMPLING A, et al: Necrotizing fasciitis. *J Laryngol Otol* 1983; 97: 193–196
29. WENIG BL, SHIKOWITZ MJ, ABRAMSON AL: Necrotizing fasciitis as a lethal complication of peritonsillar abscess. *Laryngoscope* 1984; 94 (12 pt 1): 1576–1579
30. SPANKUS EM, FLINT PW, SMITH RJ, et al: Craniocervical necrotizing fasciitis. *Otolaryngol Head Neck Surg* 1984; 92: 261–265
31. BUSH JK, GIVNER LB, WHITAKER SH, et al: Necrotizing fasciitis of the parapharyngeal space with carotid artery occlusion and acute hemiplegia. *Pediatrics* 1984; 73: 343–347
32. PACZ Z, CZAPPAN G: [Necrotizing fasciitis localized in the neck region]. *Orv Hetil* 1984; 125: 97–99
33. LEVITT GW: Cervical fascia and deep neck infections. *Laryngoscope* 1970; 80: 409–435

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Meralgia Paresthetica After Gastroplasty for Morbid Obesity

In three morbidly obese patients (mean weight 169 kg), severe hip pain developed immediately after gastroplasty. The differential diagnosis included thrombophlebitis, osteoarthritis and lumbar disc protrusion. The pattern of pain and associated numbness was characteristic of compression of the lateral cutaneous nerve of the thigh, a condition known as meralgia paresthetica. The likely cause was compression of the thigh by the metal post of the Gomez retractor. Only the most obese patients suffered this syndrome and all symptoms resolved spontaneously within 3 months.

Une douleur intense de la hanche est apparue chez trois patients souffrant d'obésité pathologique (poids moyen de 169 kg), immédiatement après une opération de gastroplastie. Le diagnostic différentiel comprenait la thrombophlébite, l'arthrose et une protrusion d'un disque lombaire. La zone d'irradiation de la douleur et de l'engourdissement qui l'accompagnait était caractéristique d'une compression du nerf cutané latéral de la cuisse, une affection connu sous le nom de méralgie paresthésique. La cause la plus probable est une compression de la cuisse par un montant de métal du rétracteur de Gomez. Seuls les patients les plus obèses ont souffert de ce syndrome et tous les symptômes ont disparu spontanément en moins de 3 mois.

Gastroplasty and gastric bypass for morbid obesity are common in North America. Patients who undergo these procedures are prone to postoperative complications including thrombophlebitis. Recently three obese patients had severe postoperative hip and thigh pain that initially was difficult to diagnose. The problem was finally determined to be meralgia paresthetica — compression of the lateral cutaneous nerve of the thigh — probably caused by the retractor used for exposure in these large patients. We describe these cases to help other surgeons who may encounter this complication of gastroplasty or gastric bypass.

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Method

Standard criteria were used to select patients for operative treatment of obesity; preoperative weight was at least 45 kg above the ideal and most were twice the ideal weight. None of the three patients (two men and one women) had preoperative hip pain or numbness. The mean age was 39 years (range from 35 to 46 years) and mean weight 169 kg (range from 136 to 208 kg).

The operations performed were Roux-en-Y gastric bypass or vertical banded gastroplasty. A Gomez retractor (Narco Scientific, Pilling Division, Fort Washington, Penn.) was used for exposure, and the patient was tilted feet down during the operation. Four vertical metal posts secure the retractor to the operating room table. The lower posts are positioned near the hips and compression may occur when the patient is wider than the table. Heparin was given subcutaneously before and after operation and ambulation was encouraged, beginning a few hours after operation.

Case Reports

Case 1

A 46-year-old man, 181 cm tall and weighing 208 kg, gave a long history of obesity and progressive sleep apnea. There was known degenerative disc disease at the L1–2 level. A gastric bypass and cholecystectomy were performed, the operation lasting 3 hours and 40 minutes. Application of the Gomez retractor was difficult because of the diameter of the hips and thighs, but the operation would probably not have been possible without it. Immediately after the operation he complained of right hip and gluteal pain, more severe than the discomfort of the abdominal incision. It was a recurrent stabbing pain, associated with anesthesia to pinprick over the lateral thigh down to the knee. During convalesence he had difficulty walking because of hip pain. X-ray films of the hip showed no local cause and an isotope venogram appeared normal. He was discharged 10 days postoperatively with persistent hip pain, which gradually subsided over 2 months although he experienced causalgia-type pain before resolution. His only major postoperative problem was hip pain, the abdominal discomfort was mild in comparison. At follow-up 11 months after operation his weight had stabilized at 117.5 kg. His hip pain and numbness had completely disappeared and the sleep apnea had resolved.

Case 2

A 37-year-old man, weighing 156 kg and 172 cm tall, underwent vertical banded gastroplasty in a 2½ hour procedure. Immediately after the operation he complained of a severe stabbing, shooting pain extending from the left hip to the knee. Analgesics intramuscularly were required to control the pain. Sensation to pinprick was decreased over the lateral left thigh and anesthesia persisted for 2 months before gradually resolving. The burning hip and thigh pain remained a problem for 2 months before disappearing completely.

Case 3

A 35-year-old woman, weighing 135.6 kg and 163 cm tall, had a vertical banded gastroplasty and cholecystectomy. The operation lasted 2 hours. The Gomez retractor was used and again was difficult to apply because the patient had very wide hips. This resulted in moderate compression of the upper thighs by the lower vertical bars of the retractor. The day after operation she experienced left hip numbness and pain, with anesthesia over the lateral thigh. Severe burning left hip and and thigh pain interfering with sleep persisted for 2 months before resolving completely. Again the hip pain was far worse than the abdominal discomfort associated with the gastroplasty. She continues to do well with a weight loss of 30 kg 6 months after operation.

Discussion

Meralgia paresthetica is the term

applied to symptoms of pain, numbness, itching or abnormal sensation in the distribution of the lateral cutaneous nerve of the thigh.[1] There is often decreased sensation over the anterolateral thigh but no motor disturbance, and men are more often affected than women. However, the discomfort is usually mild compared with that experienced by my patients.

The lateral cutaneous nerve of the thigh arises from L2 and 3. It leaves the pelvis by passing through or under the inguinal ligament medial to the anterior superior iliac spine. It passes downward on the sartorius muscle and pierces the fascia lata 10 cm distal to the anterior superior iliac spine. It appears susceptible to pressure, especially in its location near the anterior superior iliac spine or in the upper thigh. A case of meralgia paresthetica due to wearing a sword strapped to the side is cited by Stevens.[1] The mechanism of compression may be comparable to that of the Gomez retractor. The syndrome has also been described after a variety of operations,[2] although no common cause has been determined. Recently inguinal neuralgia has been described affecting the iliohypogastric, ilioinguinal or genitofemoral nerves as well as the lateral cutaneous nerve of the thigh.[3] Pain often followed pelvic or lower abdominal surgery and required operative treatment.

In my patients the severity of pain, needing strong analgesics, raised concern regarding a major orthopedic or vascular complication. The persistent burning pain is likely causalgia. Nerve section is used for chronic problems of this type but would have been difficult in these very obese patients. Nerve blocks will give temporary relief. It is likely that nerve compression was due to the lower vertical metal posts of the Gomez retractor. In all cases there was marked lateral compression of the upper thighs for 2½ to 3½ hours. Pain and numbness were noted early postoperatively. Prevention may include the use of other methods of retraction for very obese patients with wide hips, precisely the patients in whom the Gomez retractor is so helpful. Use of extra padding and placement of the lower posts well above or below the upper thigh might help to avoid the problem. In all patients the pain and numbness eventually disappeared. Successful weight loss resulted in patient satisfaction in spite of the meralgia paresthetica.

References

1. STEVENS H: Meralgia paresthetica. *AMA Arch Neurol Psychiat* 1957; 77: 557–574

2. ECKER AD, WOLTMAN HW: Meralgia paresthetica: a report of 150 cases. *JAMA* 1938; 110: 1650–1652

3. PURVES JK, MILLER JD: Inguinal neuralgia: a review of 50 patients. *Can J Surg* 1986; 29: 43–45

SESAP V Critique

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For an awake, alert, and responsive patient, only a careful history and serial physical examinations may be necessary to detect or exclude intra-abdominal injury. In the absence of adjacent associated injuries such as rib or pelvic fractures, marked tenderness and involuntary abdominal muscle spasm are considered indications for exploratory celiotomy. For this patient, evidence of circulatory instability makes delay pending further work-up unwise. Abdominal sonography, computed tomography, or selective arteriography may be useful to define the extent of injury more precisely, but are not mandatory prior to operation. Peritoneal lavage would be an unnecessary expenditure of time and money for this or any patient who has definite indications for exploratory celiotomy, such as radiologic evidence of hollow organ rupture or severe intractable hypotension in the presence of increasing abdominal distention. Peritoneal lavage is used principally for patients with equivocal findings on physical examinations and for those with impaired sensorium due to head injury, alcohol, drugs, or spinal cord injury.

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References

103/1. Bagwell CE, Ferguson WW: Blunt abdominal trauma: Exploratory laparotomy or peritoneal lavage? Am J Surg 140:368-373, 1980

103/2. Oreskovich MR, Carrico CJ: Diagnostic peritoneal lavage, in Najarian JS, Delaney JP (eds): Emergency Surgery. Chicago, Year Book Medical Publishers Inc., 1982, pp 33-41

103/3. Shires GT: Trauma, in Schwartz SI (ed): Principles of Surgery, ed 4. New York, McGraw-Hill Book Co., 1984, p 228

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Coronary Sinus Thrombosis: a Potential Complication of Right Heart Catheterization

In cardiac procedures that use access to the right atrium, the coronary sinus is at risk of accidental trauma and subsequent thrombosis as demonstrated by the two cases reported in this paper. The thromboses occurred in the setting of right heart failure and after procedures that involved catheterization or cannulation of the right atrium. A review of the literature revealed only rare instances of this complication, but, with the increasing use of the right atrium and coronary sinus for diagnostic and therapeutic purposes, injury to the coronary sinus must be considered.

Dans les opérations cardiaques nécessitant un accès à l'oreillette droite, le sinus coronarien se trouve exposé aux risques d'un traumatisme accidentel et d'une thrombose subséquente. Ce danger est illustré par les deux cas décrits dans cet article. Les thromboses sont survenues dans le contexte d'une insuffisance cardiaque droite après des interventions comprenant le cathétérisme ou la cannulation de l'oreillette droite. La littérature ne révèle que de rares cas de cette complication. Toutefois, avec un recours accru à l'oreillette droite et au sinus coronarien à des fins diagnostiques et thérapeutiques, il faut garder à l'esprit la possibilité de lésion du sinus coronarien.

The coronary sinus of the heart receives most of the cardiac venous blood and returns it to the right atrium. The right atrial ostium of the coronary sinus is large and in close proximity to right atrial orifices of the superior and inferior vena cavae. The coronary sinus is relatively unprotected and at risk of injury during procedures that use access to the right atrium, as shown in the following case reports.

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Case Reports

Case 1

A 64-year-old man who was a smoker had several previous admissions because of myocardial infarctions that resulted in refractory congestive heart failure. During two admissions he had required temporary insertion of a transvenous pacemaker and Swan–Ganz catheter respectively 9 years and 5 months previously. His death was due to large pulmonary artery thromboemboli and pulmonary infarction. At autopsy, changes of acute and chronic left and right ventricular failure were seen. There were extensive old infarcts in the left ventricle. A firm, adherent thrombus (3 cm long, 1.5 cm in diameter) occluded the coronary sinus (Fig. 1a). It had extended retrogradely into the proximal posterior interventricular vein. Other organizing mural thrombi between pectinate muscles of the posterior wall were present in the right atrium. Microscopy demonstrated a large, organizing, mural thrombus in the coronary sinus with recent occlusive thrombosis superimposed (Fig. 2A). Sections from the viable left ventricular myocardium showed an unusual amount of vascular congestion and interstitial edema.

Case 2

A 50-year-old hypertensive man, a smoker, had severe coronary artery atherosclerosis. He had undergone coronary artery bypass grafting 6 months earlier, including grafting to the left circumflex artery. He continued to smoke after that operation and had occlusion of grafts requiring regrafting. Postoperatively sepsis developed and he became hypotensive. A Swan–Ganz catheter had been inserted during his last admission. He was operated on for small- and large-bowel ischemia but remained hypotensive and died 1 week later of massive bilateral bronchopneumonia and multiorgan failure. At autopsy, a recent thrombus in the coronary sinus (Fig. 1b) and a propagating clot extending into the great cardiac vein were noted. The clot was about 5.0 cm long and 1.2 cm in diameter, again with extension into the posterior interventricular vein. Other small recent thrombi were noted on the tricuspid valve. Microscopy confirmed the recent nature of the coronary sinus thrombosis (Fig. 2B) and showed questionable congestion and edema of the myocardium.

Discussion

Coronary sinus catheterization is a potential route at cardiac surgery for retrograde perfusion of the myocardium, either for cardioplegia or for oxygenation. The sinus may also serve as a route for electrical studies and ablative procedures either operatively or by catheter. More often, the right atrium is catheterized for diagnostic and therapeutic purposes, which include placement of a central venous line and Swan–Ganz catheter and transvenous pacemaker insertions. Each of these procedures involves manipulation of the coronary venous system or the chance of accidental trauma to it. They carry the risk of subsequent occlusion, rupture, hemorrhage, throm-

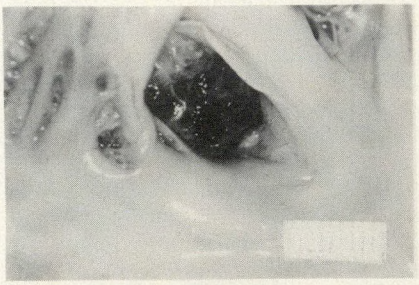

Fig. 1a

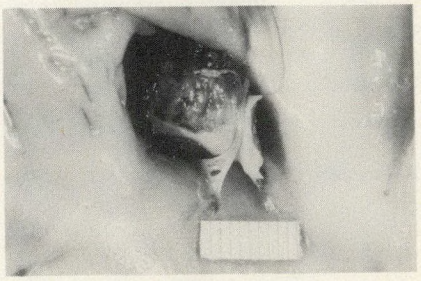

Fig. 1b

FIG. 1—Thrombi protruding from right atrial orifices of coronary sinuses in (a) case 1 and (b) case 2. Rule = 1 cm.

bosis or fibrosis of the sinus. Reports of such complications are surprisingly rare. Altman and Randolph[1] reported the case of a baby in whom a venous catheter, placed centrally for total parenteral nutrition, became wedged in the coronary sinus resulting in venous obstruction and cardiac tamponade. In a study by Fisher and colleagues,[2] one of eight patients with Wolff–Parkinson–White syndrome who underwent electrical ablation of accessory pathways through catheters placed in the coronary sinus had rupture of the sinus requiring pericardial drainage. Sinus occlusion, probably due to fibrosis, developed in two patients. Fibrosis with subsequent constriction and deformity of the orifice of the coronary sinus was also reported by Rajs[3] in 4 of 10 patients treated with transvenous cardiac pacing. In McMichael and Mousney's study[4] of 300 cases of right heart catheterization, clinical, but reversible, complications occurred in five patients whose coronary sinus was accidentally catheterized.

A review of the literature, surprisingly, yielded only two previous reports of coronary sinus thrombosis in human hearts. In each case catheterization trauma was implicated pathogenetically. Hazan and associates[5] reported on a 53-year-old woman in whom they demonstrated coronary sinus thrombosis (but only angiographically), possibly resulting from surgical trauma during mitral valve replacement or inadvertent cannulation of the coronary sinus during right heart catheterization or pacemaker insertion. Philips and colleagues[6] reported coronary sinus thrombosis as a complication of central venous catheterization for total parenteral nutrition in a 20-day-old infant.

The two patients reported here also underwent a number of invasive procedures that involved the right atrium, including transvenous temporary pacing and central venous or Swan–Ganz catheterization. The second patient also had undergone bypass grafting to the left circumflex coronary artery, with its known proximity to the coronary sinus. The operation, of course, also involves right atrial cannulation for bypass. These manipulations are all potentially traumatic and may have been important in the genesis of coronary sinus thrombosis. The finding of other atrial or tricuspid valve thrombi in each patient, of the kind usually associated with catheter trauma, supports this association. Both patients had varying degrees of left ventricular failure and superimposed pulmonary changes, contributing to low flow in the right heart, that may have favoured the thromboses. There was considerable congestion and edema of the residual viable myocardium in the first patient although available data indicate that sinus thrombosis may not be deleterious because of the extensive anastomoses between the three venous systems draining the myocardium.[2,5] Such thrombus may, however, become clinically important for other reasons, because it may embolize to lung or limit the use of the sinus for further diagnostic or therapeutic maneuvers.[5] In the future, as the coronary sinus and the right atrium are put to more diagnostic and therapeutic uses, coronary sinus pathology, including thromboses, will become increasingly frequent. These complications must be diligently sought by the clinician and by the pathologist.

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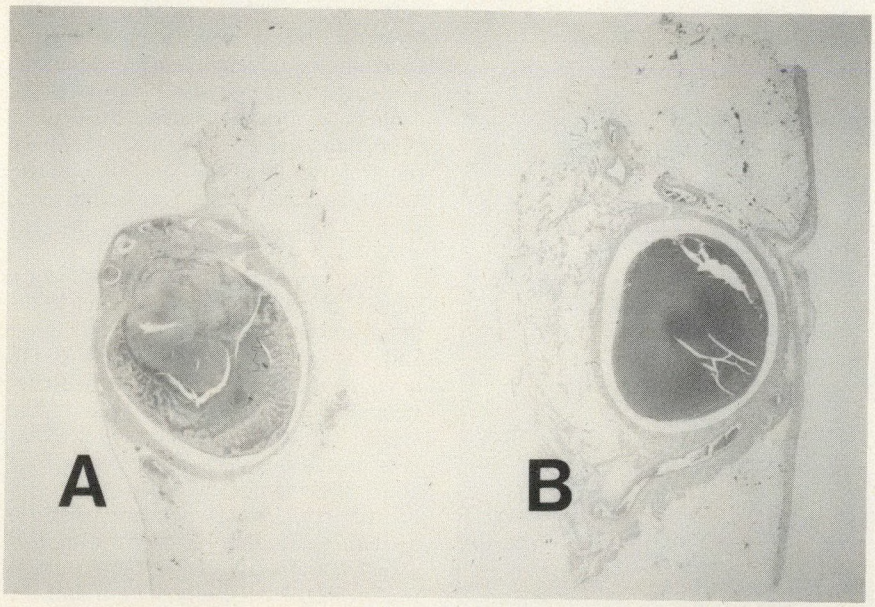

FIG. 2—Cross-section of coronary sinuses from (A) case 1 with occluding thrombus and (B) case 2 with propagated clot (hemalum-phloxine-saffron stain, original magnification × 4.5).

References

1. ALTMAN RP, RANDOLPH JG: Application and hazards of total parenteral nutrition in infants. *Ann Surg* 1971; 174: 85–90
2. FISHER JD, BRODMAN R, KIM SG, et al: Attempted nonsurgical electrical ablation of accessory pathways via the coronary sinus in the Wolff–Parkinson–White syndrome. *J Am Coll Cardiol* 1984; 4: 685-694
3. RAJS J: Postmortem findings and possible causes of unexpected death in patients treated with intraventricular pacing. *PACE* 1983; 6: 751–760
4. MCMICHAEL J, MOUSNEY JPD: A complication following coronary sinus and cardiac vein catheterization in man. *Br Heart J* 1951; 13: 397–402
5. HAZAN MB, BYRNES DA, ELMQUIST TH, et al: Angiographic demonstration of coronary sinus thrombosis: a potential consequence of trauma to the coronary sinus. *Cathet Cardiovasc Diagn* 1982; 8: 405–408
6. PHILIPS JB III, RUIZ-CASTANEDA N, SETZER ES: Coronary sinus thrombosis: a central venous catheter complication. *J Pediatr Surg* 1981; 16: 733–734

BOOK REVIEWS

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established custom, differences in equipment and also available drugs. These differences in detail may make the book less useful to the anesthetic neophyte in North America.

The most obvious examples are the different anesthesia and intensive-care-unit equipment manufactured in the United Kingdom that is not used in North America. I also believe that the chapter on intravenous therapy would have received greater emphasis and have been covered in more detail in a similar book written in North America.

There are essentially no references, and while this is a deliberate decision on the part of the authors, I think it detracts from the book, since an interested student may want more information about some of the topics covered. A few key references, to both larger standard texts and articles, could easily have been included at the end of each chapter.

The illustrations in the book are of mixed value. Line drawings and diagrams are clear and enhance the text considerably, but poorly reproduced photographs do not improve any book (and even less so when they are of equipment that is little used in North America). I find it surprising to see an illustration of a "mouth gag". Surely there are less traumatic ways of opening the mouth than with this rather barbaric tool!

A whole chapter is devoted to dental anesthesia. This may be surprising until you read that in the UK there are still more general anesthetics given for dental procedures than are given in hospitals for all other types of surgery combined. This reflects a long established and very different approach towards dental care in the UK, compared with that in North America, where far fewer general anesthetics are given for dental procedures and the techniques practised differ little from those used for any other general anesthetic.

Overall, this is a good introductory text to the practice of anesthesia. It is clearly written and easy to read and I have no hesitation in recommending it to medical students and hospital residents who wish a concise overview of this very important specialty.

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John Hunter: the First Surgical Scientist

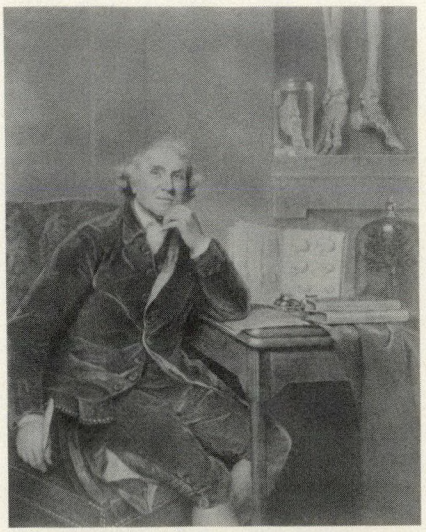
John Hunter was a brilliant surgeon and teacher, the father of scientific surgery and surgical pathology and founder of the world-renowned Hunterian museum. This essay attempts to answer the following questions. Who was this man? How did he achieve such a remarkable station? Why was he loved by some yet detested by others? When did the poor student start to become the superb teacher? How did he manage to collect the thousands of specimens he left in his museum? What is it about Hunter that makes him so well remembered today, almost 200 years after his death?

John Hunter était un brillant chirurgien et professeur, le père de la chirurgie scientifique et de la pathologie chirurgicale, fut le fondateur du musée Hunter de renommée mondiale. On tente dans cet essai de répondre aux questions suivantes. Quel homme était-il? Comment a-t-il pu atteindre une telle place dans la société? Pourquoi fut-il aimé par certains et détesté par d'autres? Quand se transforma-t-il d'étudiant pauvre en un professeur remarquable? Comment s'y prit-il pour rassembler les milliers de spécimens qu'il laissa à son musée? Comment se fait-il que, presque 200 ans àprès son décès, on se souvienne encore aussi bien de Hunter?

In the annals of surgery John Hunter is pre-eminent. His scientific curiosity, industry, skill and conceptual powers make his life an inspiration to this day. Many have written about him since his death in 1793. Most hold him in the highest esteem, but a few detractors have accused him of being uneducated, overindulged by his mother, idle until he was 20 years old, harsh, unyielding and ill-tempered in character, a poor lecturer and teacher, illiterate and lacking in classical scholarship. This brief essay is an attempt to discover the man, from his own writings and those of his family, friends and colleagues.

The Early Years (1728–1762)

John Hunter was born on Feb. 14, 1728 at Long Calderwood, near Glasgow, Scotland. The parish register states that Feb. 13 was his birthday, but Hunter celebrated his birthday on the day after. He was the youngest of 10 children, 3 of whom died in early childhood and 4 in the prime of life, most likely from tuberculosis. The three survivors were William (10 years older than John), Dorothea and John Hunter. At the age of 13, after his father died, John left school. During his childhood he is reported to have spent hours in the country studying any and all living things and asking questions that people could not answer. Like Einstein and Darwin, John did not do well in school.



John Hunter, FRS (1728–1793).

At the age of 17 years he moved to Glasgow to live with his sister and her husband. At the age of 20, he moved to London to work with his brother William, an established anatomist and obstetrician. John became skilled at dissection. He studied anatomy from cadavers instead of books. He made friends with owners of menageries, helped feed the animals and bought their carcasses for dissection. Friends informed him of the bodies of human and animal freaks he could dissect. He was liked by the "body snatchers" so he received preferential treatment.

During this time he started an apprenticeship in surgery under Cheselden at the Royal Hospital, Chelsea, and after Cheselden's death under Percivall Pott at St. Bartholomew's Hospital, London. In 1753, William Hunter became master of anatomy at Surgeon's Hall; this enabled John to continue his dissecting and to help his brother in teaching anatomy. However, his interest in physiological function was greater than in structural anatomy. In 1759, after 11 years of experience in anatomical dissection, "inflammation of the lungs", possibly tuberculosis, developed. In 1760, near the end of the Seven Years' War he had recovered sufficiently to join the army. He was 32 years old.

There are three likely reasons why Hunter joined the army — to earn a livelihood, to obtain a medical qualification and to gain surgical experience. He attended the wounded and sick at Belle-Île-en-Mer and in Portugal, noting the stages of inflammation and factors that promoted healing. His surgical knowledge and ability were ahead of his time, and he freely criticized his contemporaries. He used his spare time for observation and experimentation, studying the regeneration of lizard tails, hibernation and the hearing of fish. He brought back over 200 specimens, many of which are still in the

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Hunterian Museum. Appreciating the significance of this experience, Hunter wrote:

> In the year 1761 I had the honour of being appointed by your Majesty a Surgeon on the Staff in the expedition against Belleisle ... gave me extensive opportunities of attending to gunshot wounds, of seeing the errors and defects in that branch of military surgery, and of studying to remove them. It drew my attention to inflammation in general and enabled me to make observations which have formed the basis of the present Treatise...[1]

The Later Years (1763-1792)

In 1763 he returned home, now a surgeon, but without a hospital appointment. To earn a living he worked in Spence's dental surgery and slowly built a surgical practice. Later he started a school to teach anatomy and surgery. He bought land at Earl's Court, two miles from London, and built a house where he kept eels, fish, leopards, jackals, buffalo, stallions, sheep, goats, an ostrich, bees and silkworms. Here, and in his successive London houses, he dissected, wrote, dictated notes, saw patients and housed pupils.

In 1767 he was elected a fellow of the Royal Society, and the following year was admitted a member of the Company of Surgeons of London (which became the Royal College of Surgeons in 1800). A year later he was appointed surgeon to St. George's Hospital, a post he held until his death.

It is interesting that Hunter was accused of being uncultured, ill-bred and uncouth, yet he was on excellent terms with prominent figures of his day and held a respected place in society. At his election to the staff of St. George's Hospital in 1768, he received 114 out of 161 votes. As to being a poor lecturer and teacher, Hunter attracted many students, in fact more than all other surgeons at St. George's Hospital for 25 years. Among his pupils were such distinguished surgeons as Astley Cooper, John Abernethy and Charles White and such eminent physicians as Edward Jenner and James Parkinson. Notes of his lectures taken by students were copied, shared and preserved. One student, Henry Cline, wrote:

> When I was only 24 years of age I had the happiness of hearing the first course of lectures which John Hunter delivered. I had been at the time for some years in the profession and was tolerably well acquainted with the opinions held by surgeons most distinguished for their talents then residing in the Metropolis; but having heard Mr. Hunter's lectures on the subject of disease, I found them so far superior to everything I had conceived or heard before, that there seemed no comparison between the great mind of the man who delivered them and all the individuals, whether ancient or modern, who had gone before him.[2]

In 1771, at the age of 43 years, Hunter married Anne Home, who was attractive, intelligent, talented in both music and poetry, a splendid hostess and a devoted wife. They had four children, two of whom died in childhood. Hunter received great support from his wife and wrote:

> As to myself, with respect to my family, I can only say that I am happy in a wife ... it appears to be Anny's enjoyment in seeing me pleasing myself: while all these concurring circumstances go on, I must continue to be one of the happiest men living...[3]

In 1776 Hunter was appointed surgeon extraordinary to King George III. In 1781 he helped found the Royal Veterinary College and became the first vice-president. In 1786 he received the Copley Medal, the highest award of the Royal Society and was appointed assistant surgeon-general to the armed forces; in 1790 he became the surgeon-general and inspector-general of regimental hospitals.

During this period Hunter was actively engaged in establishing his museum. Existing museums were simply collections of curiosities and if organized at all were based on the rigid concept of the immutability of species supported by Linnaeus and Cuvier. Hunter designed his museum "to demonstrate, in terms of anatomical preparations, how function determined structure, how a like physiological goal might be attained by diverse morphological arrangements and how the morphological ... provided the key to understanding of function and behaviour".[4] He arranged the specimens in three main groups: to demonstrate structures developed for the preservation of the individual; those for ensuring the continuity of the species; and the third a group of pathological specimens. He also had about 3000 animal and vegetable fossils. Hunter recognized the value of his museum and requested that it be preserved as an entity following his death. The Hunterian Museum is "one of the most remarkable monuments that any man has ever raised to perpetuate his memory ... it is John Hunter's greatest unwritten book".[5]

His Death

On Oct. 16, 1793, Hunter died during a stormy meeting in the board room of St. George's Hospital. He was buried at the Church of St. Martin-in-the-Fields. In 1859 he was reinterred at Westminster Abbey.

Although it is known that he experienced repeated attacks of angina pectoris, starting in 1773, factors leading to Hunter's death remain in dispute. The allegation that he inoculated himself with syphilis has been publicized widely since 1925, when Sir D'Arcy Power gave a Hunterian Oration entitled, "John Hunter: A Martyr to Science". Power claimed that Hunter originally inoculated himself and that he "died of syphilitic disease of the arterial system, and ..., in addition to the angina pectoris due to this cause, he suffered for many years from cerebral syphilis".[6]

Qvist[7] has studied this matter extensively and has come to the conclusion that "the whole idea of Hunter inoculating himself with a venereal disease is preposterous". It is not supported by the autopsy findings, the clinical features of his illness or by Hunter's own description of the inoculation experiment performed in 1767. Hunter's autopsy showed advanced generalized atherosclerosis with calcified coronary and internal carotid arteries. The clinical features of his illness were those of myocardial and cerebral ischemia. Throughout his works, Hunter stated quite clearly whenever he was the subject, yet his own account of the inoculation experiment does not identify the subject and does not state that he inoculated himself, so it is unlikely that this would be the only occasion he would not do so.

Hunter's Character

Another controversial issue arising since Hunter's death concerns his character. Although many uncomplimentary statements have been made, it seems that people who knew him well admired him. Everard Home,[8] Hunter's brother-in-law, wrote:

> Mr. Hunter was of a short stature, uncommonly strong and active, very compactly made and capable of great bodily exertion. His countenance was animated, open, and in the latter part of his life deeply impressed with thoughtfulness ... His temper was very warm and impatient ... He hated deceit, and ... detested it in others, and too openly avowed his sentiments ... In private practice he was liberal, scrupulously honest in saying what was really his opinion of the case, and ready upon all occasions to acknowledge his ignorance ... Public-spirited to an extreme, he valued money no farther than as it enabled him to prosecute and extend his various, and nearly universal, researches...

Adams, who knew him well, stated,[9] "Mr. Hunter's manners were extremely companionable. His wit, or more properly his archness, was always well directed." Holland[10] wrote, "John Hunter was neither polished in his manner nor refined in his expression, but from originality of thought and earnestness of mind he was extremely agreeable in conversation; ... His countenance was indeed full of genius..."

William Clift, an apprentice and a devoted amanuensis to his master for the

last 2 years of Hunter's life, wrote that Hunter was

> generally, though cheerfully, taciturn — many a morning's labour passing over with scarcely a word of discourse: but shrewd and witty in his remarks ... mild and kind in his manner, sufficiently but not servilely, courteous to everybody, and made no distinction between high and low, great or small; spoke as kindly and familiarly to his gardener or myself as to his equals or superiors...[11]

Clift also wrote that he "could relate a professional anecdote very humerously, concisely and with much point, but with never the slightest inclination to ill-natured remarks, swearing or obscenity for which many of his contemporaries were notorious."[11]

Hunter had a degree of humility that went unrecognized by many. Qvist[7] wrote, "In spite of criticisms that John Hunter was arrogant and abrasive, there are numerous passages in his writings that show he was self-critical in his work and that he had that innate modesty and profound humility that are essential in the search for truth in nature."

Hunter freely admitted his failures when he recognized them. On one such occasion he wrote:[12] "Whenever I have seen the dura mater opened, ... the patients have died. This was the case with a Mr. Cooper, whose dura mater I opened ... he died, and I think it is probable I killed him by opening the dura mater."

Another great disappointment to him followed his unsuccessful attempts at freezing and reviving animals:

> Till this time I had imagined that it might be possible to prolong life to any period by freezing a person in the frigid zone, as I thought all action and waste would cease until the body was thawed. I thought that if a man would give up the last ten years of his life to this kind of alternate oblivion and action, it might be prolonged to a thousand years; and by getting himself thawed every hundred years, he might learn what had happened during his frozen condition. Like other schemers, I thought I should make my fortune by it; but this experiment undeceived me.[13]

His Scholarship

John Hunter has also been accused of being illiterate and lacking classical scholarship. He spent most of his time on original research, but he did use references and had quite an extensive library. He wrote to the Company of Surgeons in 1786 complaining about the absence of a library and stating that it was essential to acquire one. Hunter published numerous scientific papers in the *Transactions of the Royal Society*. Since most of his work was original he had to create many new words, especially in surgical pathology.

Hunter's field of interest was broad, covering the whole of the natural sciences. His works are all based on his own original research. As well as numerous papers published in the *Transactions of the Royal Society*, Hunter published three books: *Treatise on the Natural History of the Human Teeth* (part I in 1771 and part II in 1778), *A Treatise on the Venereal Disease* (1786) and *Observations on Certain Parts of the Animal Oeconomy* (1786). Hunter prepared the manuscripts of three more books that were published posthumously: *A Treatise on the Blood, Inflammation and Gun-shot Wounds* (1794); *Observations and Reflections on Geology* (1859) and *Memoranda on Vegetation* (1860). He also left numerous manuscripts, notes and lectures recorded by students.

Hunter's curiosity about all facets of nature involved commitments from those who knew him. When Hunter was interested in the relationship of fat with temperature and hibernation he elicited the cooperation of Edward Jenner. During this particular study, Jenner was experiencing serious personal problems. In a letter to Jenner, Hunter wrote:

> I own I was at a loss to account for your silence, and I was sorry for the cause. I can easily conceive how you must feel, for you have two passions to cope with, viz., that of being disappointed in love, and that of being defeated; but both will wear out, perhaps the first soonest, I own I was glad when I heard you was to be married to a woman of fortune; but 'let her go, never mind her'. I shall employ you with hedgehogs... I want you to get a hedgehog in the beginning of winter, and weigh him; put him in your garden and let him have some leaves, hay, or straw to cover himself with, which he will do, then weigh him in the spring and see what he has lost...[13]

It is not feasible in an overview such as this to give an adequate sampling of Hunter's works. However, it would be improper not to include a glimpse of the spectrum of his writings, so I have selected a few of his quotes on a variety of medically related topics. I make no apology for the lack of cohesion between the quotes chosen, as Hunter wrote on such varied phenomena.

> Bone is not the original skeleton in any animal, but only of the adult; for in the first formation of any animal, which afterwards is to have bone, the skeleton is either membrane or cartilage, which is changed for bone, but not into bone.[13]

> The teeth are adapted to the dividing and masticating or grinding the food proper for the animal, but besides all this, are adapted to the catching of it, as in many fish and wild animals. Teeth also are in many a defence from enemies, and seem entirely given for this purpose, as in the tusk of the elephant.[13]

> There are very few dead bodies in which the stomach is not, at its great end, in some degree digested ... It struck me that it was from the process of digestion going on after death, that the stomach, being dead, was no longer capable of resisting the powers of that menstruum, which itself had formed for the digestion of its contents. These appearances of the stomach after death, throw considerable light on the principles of digestion, and show that it neither depends on a mechanical power, not contractions of the stomach, nor on heat, but something secreted on the coats of the stomach and thrown into the cavity, which there animalises the food, or assimilates it to the nature of the blood.[13]

> The other part of this system, called lymphatics, though long known, was not in the least suspected of performing the operation of absorption, but they were still supposed to be continuations of the extreme ends of arteries, which were not large enough to carry blood, only carrying the serum or lymph; but from their similarity to the lacteals, which were now known to be absorbents, it became at last plain and evident to common sense they must also absorb.[13]

> ...the heart exerts its influence upon the different parts of the body in proportion to their vicinity to it; and the more distant that the parts are, the weaker are their powers ... In diseases we see mortification arising...in the extremities oftener than in other parts, ... the heart not propelling the blood to these distant parts with equal force.[14]

> ...an artery ... had two powers, the one elastic and the other muscular ... we must suppose the elastic best fitted for sustaining a force applied to it, such as the motion of the blood given by the heart, and propelling it along the vessel; the muscular power most probably is required to assist in continuing that motion, but certainly was intended to dispose of the blood when arrived at its place of destination.[13]

> ...the use of fat or oil in an animal body would appear to be of three kinds: nourishment, production of heat, and retention of heat.[13]

> The bodies called kidneys are glands intended for a secretion of a fluid, which in common language is called urine. Their use is immediately to carry out of the circulation such parts as are useless and obnoxious...[13]

Hunter studied deer antlers to learn that new bony growth produces new vascularity and noted that "the external carotids in the stag, when his horns are growing, are much larger than at any other time".[1] He also ligated the carotid artery of a deer, noted the changes in the antler and the development of collateral circulation. He concluded that intrinsic vessel disease must be present for aneurysms to form, so it would be important to ligate an artery at a distance from an aneurysm. This led to several successful operations of popliteal artery aneurysm by ligating the femoral artery proximally. Perhaps his greatest contribution to surgery was his repair of aneurysms, thereby preserving the limbs

of many a soldier who would otherwise have had an amputation.

Hunter described three main types of inflammatory reaction; adhesive, suppurative and ulcerative. Recognizing its importance in pathology he wrote:[12] "The operation in the body called inflammation is one of the most common and most extensive in its effects ... producing abscesses, fistulas, diseased bones, etc., and in many diseases is the first step towards a cure; so that it becomes a first principle in surgery."

John Hunter's masterwork, *A Treatise on the Blood, Inflammation and Gunshot Wounds*, shows that he understood the pathology of gunshot wounds and the three most important factors contributing to wound morbidity: contamination from the exterior, an impaired blood supply and the presence of devitalized tissue. Qvist[7] believed that "If Hunter's treatise on gunshot wounds had been more carefully studied instead of being so frequently criticized, perhaps the logical operation of excision of wounds of violence might have been evolved before Milligan's time."

Hunter's Legacy

In 1799 the British government decided to purchase the Hunterian Museum for the nation, giving custody to the Company of Surgeons, which was renamed and reconstituted as the Royal College of Surgeons in 1800. According to Qvist:[7]

> The acquisition of custodianship of the Hunterian Collection was one of the most important in the history of the Royal College of Surgeons of England because, during the early years of its development, the College gained far more credit, both at home and abroad, from the excellence of the Museum than from the activities of the surgeons.

William Clift is credited with preserving the integrity of the museum. He was appointed conservator until he retired in 1842. His son-in-law Sir Richard Owen succeeded him. The efforts of these two greatly enhanced the reputation of the Hunterian Museum both at home and abroad.

John Hunter is remembered for raising surgery from a craft to a science and for developing the world's finest museum of comparative anatomy and surgical pathology. He dissected thousands of human bodies and hundreds of different species of animals. His emphasis on observation, experimentation and clinical application have led him to be accepted as the founder of surgical pathology and scientific surgery. The Hunterian Museum, John Hunter's unwritten book, is a lasting monument containing 14 000 specimens. He was once described[3] as "anatomist, biologist, naturalist, physician, surgeon and pathologist, all at once and all in the highest". His influence continues to pervade English-speaking medicine and surgery, as testified by the numerous lecturers, biographers and researchers who have studied his life and work. The Hunterian orations, which began in 1914, continue to this day to perpetuate his contributions to medicine and science. His presence is even felt 200 years later and 2000 miles away at the University of Calgary medical school where we continue to pay tribute to this great man annually. Hunter himself, with his constant emphasis on scientific reasoning, would probably most appreciate being remembered as the world's first surgical scientist.

I thank Dr. Peter Cruse for his stimulation to research the life and contributions of Hunter.

References

1. HUNTER J: A treatise on the blood, inflammation, and gun-shot wounds. In PALMER JF (ed): *The Works of John Hunter*, vol III, Longman, London, 1983 (reprint of 1837 edition): 9
2. CLINE H: *Hunterian Oration*, 1824
3. PAGET S: *John Hunter*, T. Fisher Unwin, London, 1897
4. CAVE AJE: Wood Joines Medal Lecture, 1978. A constellation of conservators. *Ann R Coll Surg Engl* 1980; 62: 66–70
5. JONES WF: John Hunter's unwritten book. *Lancet* 1951; 2: 778–780
6. POWER D: *Hunterian Oration 1925. John Hunter: A Martyr to Science in Selected Writings 1877-1930*, Clarendon Pr, Oxford, 1931: 1–28
7. QVIST G: *John Hunter 1728-1793*, Heinemann, London, 1981
8. HOME E: A short account of the author's life. In HUNTER J: *A Treatise on the Blood, Inflammation and Gun-shot Wounds*, G. Nichol, London, 1794
9. ADAMS J: *Memoirs of the Life and Doctrines of the Late John Hunter, Esq., Founder of the Hunterian Museum, at the Royal College of Surgeons in London*, J. Callow, London, 1817
10. HOLLAND HR: *Further Memoirs of the Whig Party 1807–1821*, J. Murray, London, 1905
11. DOBSON J: *William Clift*, Heinemann, London, 1954
12. HUNTER J: Lectures on the principles of surgery. In PALMER JF (ed): *The Works of Hunter*, vol I, Longman, London, 1835
13. ALLEN E: *Hunterian Museum Guide*, 1974
14. HUNTER J: A treatise on venereal disease. In PALMER JF (ed): *The Works of John Hunter*, vol II, Longman, London, 1835

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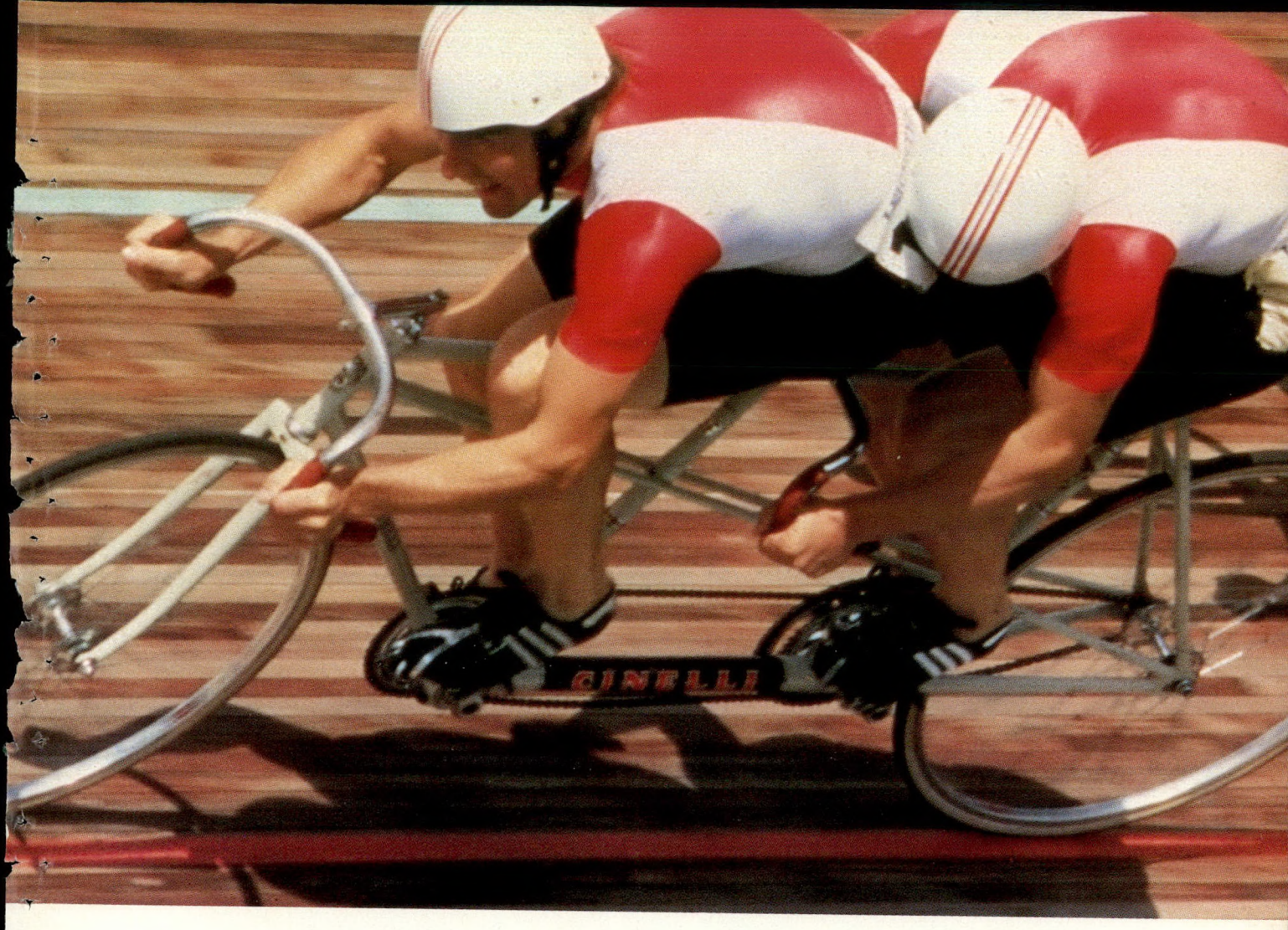

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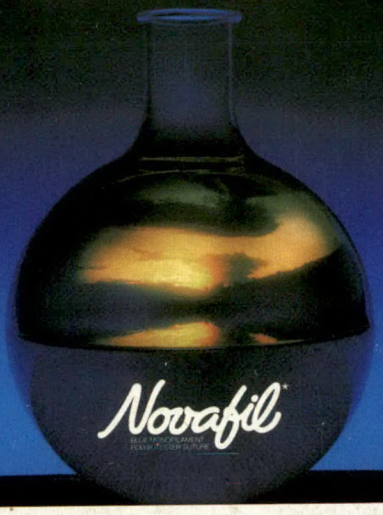

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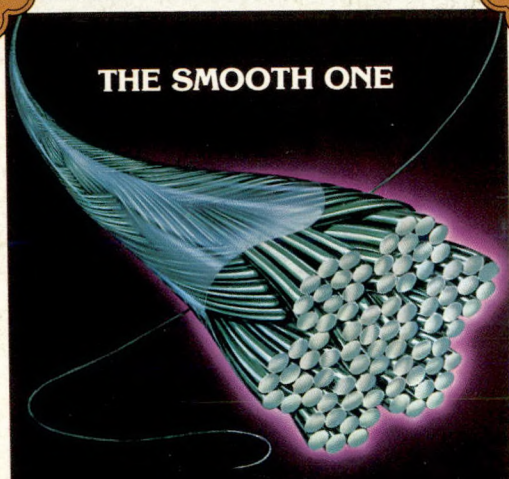

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